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PATHOGENESIS AND TREATMENT OF CHOLESTEATOMA IN CHRONIC SUPPURATION OF THE TEMPORAL BONE

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The well known dangers of a suppuration of the middle ear due to cholesteatoma can reliably be avoided by the so-called radical mastoidectomy. However, many patients are disappointed by the result of the operation because of the frequently persisting annoying discharge and continued loss of auditory capacity despite complete removal of the cholesteatoma. It seems that today better operative results can be achieved because surgical advances, made since the advent of fenestration surgery, can be employed on the basis of an exact knowledge of the pathogenesis of cholesteatoma. This very point, however, is unfortunately the subject of widely differing opinions. For the establishment of a better therapy of cholesteatoma a discussion of its pathogenesis is therefore necessary.

Many textbooks contain the statement, only recently reconfirmed by Marcus Diamant, that a chronic otitis media develops as a rule within a mastoid which is relatively devoid of cells and is due to a sclerosis of the mastoid bone. According to the opinion of Wittmaack, this predisposing sclerosis of the mastoid is caused by acute infections of the middle ear in the newly born.

From the Clinic of Oto-Rhino-Laryngology, Zürich University (Prof. L. Rüedi, M.D., Director). Read at the Sixth International Congress of Otolaryngology, Washington, D. C., May 6, 1957.

In an extensive histological material we have examined the normal development of the air spaces of the middle ear and the effects exerted thereon by infections of the middle ear at an early infantile age, in order to check the teachings of Wittmaack. There is evidence that the development of the middle ear air spaces has many more variations than assumed by Wittmaack. Already at the time of birth there are distinct individual differences in the development of cells. In addition to the main air spaces, certain new-borns possess numerous cells in the temporal bone and in the growing mastoid process. In others of the same age there is the antrum only. Moreover, the development of the air spaces is more complicated than described by Wittmaack. In the embryonic stages the formation of the middle ear spaces proceeds in two different ways. In a first process of development the main spaces of the middle ear develop in a fibrous bony mesenchyme and in about the sixth fetal month a widely varying anlage of cells starts from the antrum into the growing bone. These cells are completely filled with loose embryonic connective tissue. In a second process of development these middle ear spaces preformed in the bone are pneumatized by an ingrowing strand of mucous membrane coming from the eustachian tube. The mucous membrane expands within the preformed cells at the expense of the slowly shrinking connective tissue. This process of pneumatization also shows individual variations of speed. Accordingly, several possibilities of combination between the individually varying preformation of spaces and their pneumatization which also proceeds at an individual pace will be found at the time of birth. Four of the many combinations possible are shown in Figure 1.

In this treatise we are interested in those new-borns whose system of cells is well preformed but not yet or only incompletely pneumatized. As a rule the epitympanic recess, and especially Prussak's space, is filled with submucous connective tissue which is in direct contact with Shrapnell's membrane. Because of these strands of submucous connective tissue the mucous membrane appears to be hyperplastic but this is not due to an inflammatory agent in Wittmaack's sense.

These strands of submucous connective tissue normally (i.e., damaging influences lacking) disappear during the first year of life. Experience shows that infants with this congenital "connective tissue hyperplasia" are especially susceptible to infectious inflammations of the middle ear. According to our observations the development of spaces is thereby influenced in the following manner:

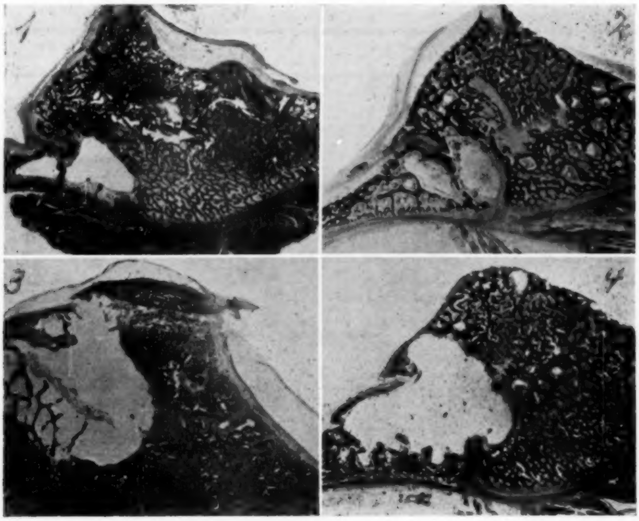

Fig. 1.—These sections run vertically at the identical plane through the antrum of four newly-borns without ear pathology. In Case 1 only a small antrum has developed which is still filled by loose connective tissue. In Case 2 the equally small antrum is pneumatized. In Case 3 there is a large antrum with cells originating from it. In all these spaces remaining loose connective tissue is found. Case 4 demonstrates a large antrum. Its rather numerous cells are almost completely pneumatized.

The banal acute otitis of the newborn effects a transitory arrest of pneumatization whereas the preformation of the bony cavities usually continues undisturbed. The sclerosing effect of the acute otitis of the newborn on the bony middle ear as stipulated by Wittmaack cannot be found in our material. The necrotizing otitis of the newborn, however, which regularly occasions large perforations of the tympanic membrane affects both phases of the space formation in a definite manner. Within already existing cells pneumatization is arrested and the phase of preformation of bony cells even switches to the contrary. The already existing cells which still contain connective tissue are replaced by sclerotic bone through infectious ossification of their contents. In order to prove that the relative lack of cells and the bony sclerosis of the mastoid constitute a result rather than a cause of infantile otitis media we have communicated earlier, comparing histological examinations of 11 cases of unilateral chronic

TABLE I

NO. INIT.	AGE	MIDDLE EAR	MARGINAL PERFORATION	BONE CAVITY-FORMATION	PNEUMATIZATION
1 Z. R. ♂	4 WEEKS	R: OT. MED. NECROT. CHOL.	LARGE	EXTENSIVE	INCOMPLETE
		L: OT. MED. NECROT. CHOL.	LARGE	EXTENSIVE	INCOMPLETE
2 M. M. ♀	16 MONTHS	R: NORMAL		EXTENSIVE	COMPLETE
		L: OT. MED. TBC.	LARGE	EXTENSIVE	MASTOIDIT. TBC.
3 H. W. ♂	11 MONTHS	R: OT. MED. NECROT. CHOL.	SMALL	EXTENSIVE	NEARLY COMPLETE
		L: OT. MED. NECROT. CHOL.	LARGE	EXTENSIVE	MASTOIDITIS AND OSTEOMYELITIS
4 M. M. ♀	4 YEARS	R: OT. MED. TBC. BEGINNING CHOL. L: ?	SMALL	EXTENSIVE	INCOMPLETE
5 B. T. ♂	5 YEARS	R: OT. MED. NECROT. CHOL.	LARGE	EXTENSIVE	COMPLETE
		L: OT. MED. ACUTA		EXTENSIVE	COMPLETE
6 ST.- ♂	11 YEARS	R: OT. MED. CHRON.	LARGE	EXTENSIVE	INCOMPLETE
		L: NORMAL		EXTENSIVE	INCOMPLETE
7 K. H. ♂	14 YEARS	R: OT. MED. CHRON. (CENTRAL PERF.)		EXTENSIVE	INCOMPLETE
		L: OT. MED. CHRON. CHOL.	LARGE	EXTENSIVE	INCOMPLETE
8 SCH. G. ♂	15 YEARS	R: OT. MED. CHRON. CHOL.	LARGE	EXTENSIVE	INCOMPLETE
		L: ?			
9 K. M. ♀	16 YEARS	R: OT. MED. CHRON. CHOL.	LARGE	MODERATE	INCOMPLETE
		L: ?			
10 B. G. ♀	19 YEARS	R: ?			
		L: OT. MED. CHRON. CHOL.	LARGE	MODERATE	INCOMPLETE
11 G. K. ♀	22 YEARS	R: OT. MED. CHRON. CHOL.	LARGE	MODERATE (SCLEROSIS)	VERY INCOMPLETE (NEW BONE FORMATION)
		L: OT. MED. CHRON. CHOL.	LARGE	POOR (SCLEROSIS)	VERY INCOMPLETE (NEW BONE FORMATION)
12 K. F. ♂	23 YEARS	R: NORMAL			
		L: OT. MED. CHRON. CHOL.	SMALL	POOR (SCLEROSIS)	INCOMPLETE (NEW BONE FORMATION)

Tympanic membrane findings and histologically controlled cavity formation of the middle ear in 12 adolescent cases of cholesteatoma of the middle ear.

otitis media. In the ear affected by chronic suppuration a definite narrowing of the middle ear spaces and sclerosis of the bone were demonstrated, whereas the normal side showed completely pneumatized cells of normal variation in size, in bone of spongy and lamellar structure.

Further proof of our heretic opinion is moreover found in the histological specimens of 12 youthful cases of cholesteatoma which are compiled in Table I. Despite their chronic suppuration of the middle ear 8 patients between 8 weeks and 15 years of age still show an extensive cell system in the mastoid. But these cells are incompletely pneumatized; they still contain connective tissue. Four further cases

TABLE II

AGE	EXTENSIVE TO MODERATE	SLIGHT TO LACKING
5 – 10 YEARS (32)	55 %	45 %
11 – 15 YEARS (39)	44 %	56 %
16 – 20 YEARS (28)	21 %	79 %
21 – 25 YEARS (42)	12 %	88 %

Air cell formation of the mastoid in 141 radically operated adolescent cases.

between the age of 15 and 23 years show evidence of definite arrest of space formation in the sclerotic bone on the affected side. In the connective tissue contained therein there are regularly signs of inflammatory ossification. These histological observations are in agreement with the often surprising findings in mastoids of infants which were radically operated upon. In these cases the x-ray films frequently fail since the cell cavities still contain inflamed connective tissue and the thin bony cell walls yield no x-ray contrast. Among our radically operated 141 youthful cases (Table II) we found an extensive to moderate stage of space development in the mastoid in 55 per cent of those between the ages of 5 and 10, and only in 12 per cent between 21 and 25 years of age. Therefore the older a patient afflicted with cholesteatoma is, the scantier the cells in the mastoid continuously undergoing sclerosis.

On the basis of histological controls and surgical findings we therefore arrive at the conclusion that the relative lack of cells and sclerosis of the mastoid which are typical of chronic otitis media do not constitute a cause but a result of the chronic suppuration of the middle ear usually starting in early infancy. Infants who do have a well preformed anlage of the middle ear space system only lacking in its pneumatization are predisposed to the inception of a chronic otitis media with or without formation of a cholesteatoma, because infections show a special affinity to the "hyperplastic" mucous membrane filling these cavities.

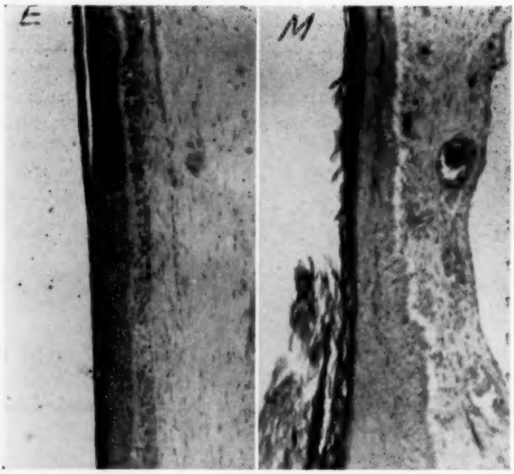

Fig. 2.—A histological section through the epidermis of the external auditory canal (E) and a section of the matrix of a cholesteatoma (M).

A further question remaining unsettled concerns the *structure of the matrix of the cholesteatoma.*

According to Marx the matrix and the epidermis are of identical structure, the matrix lacking only glands and hair. In comparing a histological section through the epidermis (E) of the external auditory canal with a corresponding section of the matrix (M) of a cholesteatoma (Fig. 2) superficially desquamated horn lamellae and several layers of stratified squamous epithelium will be found in both specimens. Below, there is in both sections a regular, basally arranged layer of membraneless columnar cells with oblong nuclei. This layer of basal cells is distinctly defined from the connective tissue base. The epidermal prickle cells and the basal cells of the matrix are identical in all their histological criteria. The matrix of the cholesteatoma shows therefore the same structure as the skin as described by Marx.

An additional question is: *How and where does a cholesteatoma develop and what is the mode of growth of its matrix?*

According to Diamant and in agreement with Teed, the cholesteatoma develops behind an initially intact tympanic membrane due to

Fig. 3.—Vertical section of a Shrapnell's cholesteatoma in the right ear of a 23 year old man.

an inborn dislodgement of squamous epithelium in the mucous membrane of the middle ear. Why cholesteatoma germs begin to grow is not discussed by Diamant and to my knowledge this author has not examined the growth of the matrix of the cholesteatoma.

In the Anglo-saxon literature an infectious metaplasia of the mucous membrane of the middle ear is, as a rule, considered to be the cause of a cholesteatoma. The cuboidal epithelium of the epitympanic recess is said to be transformed into the stratified squamous epithelium from which the cholesteatoma originates. The cholesteatoma thus originated grows because of congestion of its horny lamellae according to Tumarkin.

In the German-speaking countries, Link's idea of a mucous membrane metaplasia has been rather generally abandoned in favor of that of an inflammatory immigration of stratified squamous epithelium from the cutaneous auditory canal into the middle ear after Habermann successfully demonstrated this process histologically in the course of a necrotizing otitis media and the subsequent development of a cholesteatoma. The adherents of this immigration theory, Bezold, Manasse, et al., subsequently consider the invasion of the epithelium of the external auditory meatus as an attempt at healing which replaces the damaged mucous membrane of the middle ear.

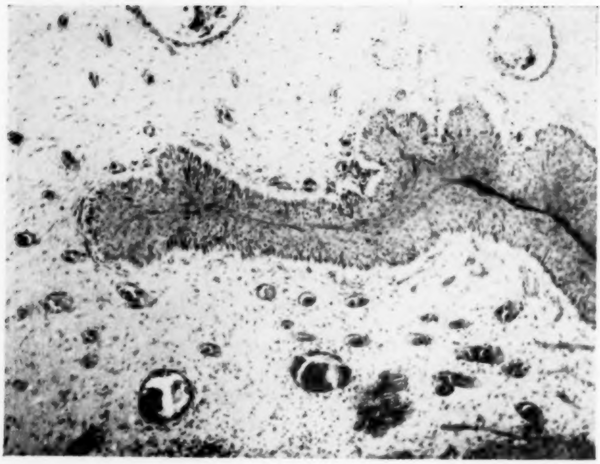

Fig. 4.—Higher magnification of Figure 5: A cholesteatomatous cone of basal cells infiltrating the connective tissue.

In the majority of cases this healing process is said to fail, due to the unfavorable conditions of space (Schwarz). The epidermis having invaded the middle ear spaces is subject to a humid and warm climate. Thus irritation occasions a high degree of desquamation and the epidermis is changed into the matrix of a cholesteatoma. Under the pressure of the congested masses of the cholesteatoma, which is frequently secondarily infected the surrounding bone undergoes osteoclastic lysis. It is said that the surface of the matrix can now expand passively more or less as demanded by the increase in lamellae of the cholesteatoma.

In the clinically especially important group of cholesteatomas with small superior marginal perforations, however, the classical immigration theory encounters certain difficulties. In these cases the disease, lacking symptoms almost completely, commences without evident connection with an inflammation of the middle ear. When the small perforation of the tympanic membrane is finally discovered a cholesteatoma has as a rule already developed in the epitympanic recess. Exceptionally there is a cholesteatoma but no perforation of the tympanic membrane. For this reason Diamant considers the immigration of stratified squamous epithelium from the external auditory meatus an impossibility.

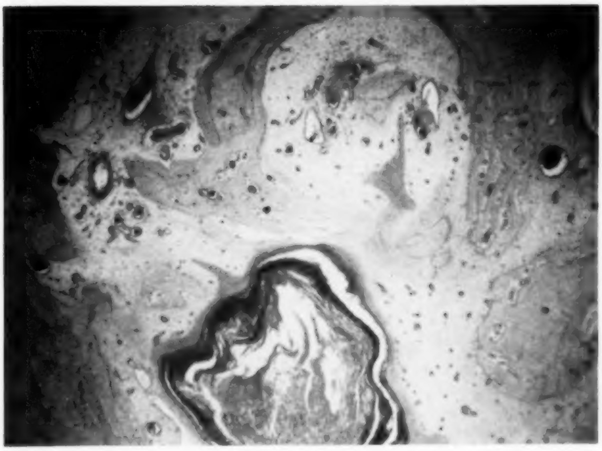

Fig. 5.—Further detail of Figure 3: The behavior of the actively growing matrix towards the surrounding bony tissue and the reaction of this bone.

Nevertheless Lange, Albrecht, Steurer, Saxén et al. hold that the invasion of epidermis of the external auditory canal is responsible for this type of cholesteatoma also, being due to an active proliferation of the basal cells in Shrapnell's membrane into the middle ear which is induced by inflammatory irritation. The cholesteatoma thus established is said to invade the auditory canal only secondarily after the occurrence of a small superior marginal perforation. This process, however, shows no resemblance to an attempt at healing. There is no lesion of the mucous membrane which has to be covered and behind the initially intact tympanic membrane, which only later shows a defined perforation, the cholesteatoma seems to grow more actively and aggressively than those cholesteatomas with a large defect of the tympanic membrane which appear to pursue a less malignant course.

Thus the only general agreement among the several authors is that development of a cholesteatoma as a rule is due to inflammations of the middle ear. The question remains open whether cholesteatoma is due to an inborn dislodgement of squamous epithelium present in the mucous membrane, or to a metaplasia of the mucous membrane of the middle ear, or to immigration of epidermis of the external auditory meatus. To my knowledge neither the germ dislodgement postulated by Teed nor the inflammatory metaplasia of the mucous

Fig. 6.—Otitis media chronica cholesteatomatosa with a large superior marginal perforation in the case of a 15 year old boy.

membrane have been found histologically and we have searched for these changes in vain in serial histological sections of 150 normal and pathological ears. At best the germ dislodgement which is theoretically possible (Mündnich) and the mucous membrane metaplasia respectively constitute rarities, whereas cholesteatomas of the middle ear occur frequently.

The structure of the bronchial mucous membrane used for comparison is much more differentiated than the mucous membrane of the middle ear, and under normal condition patches of transition to squamous, metaplastic epithelium can be found in this ciliated columnar epithelium. This important requisite to stratified squamous epithelium metaplasia is lacking in the mucous membrane of the middle ear.

In order to check the immigration theory we will now examine 12 adolescent cases of cholesteatoma with small and with large superior marginal perforations and we will observe the mode of growth of the matrix.

In the group of four cholesteatomas of Shrapnell's membrane with small marginal perforation evidence of active infiltrative growth as described by Lange et al. is found.

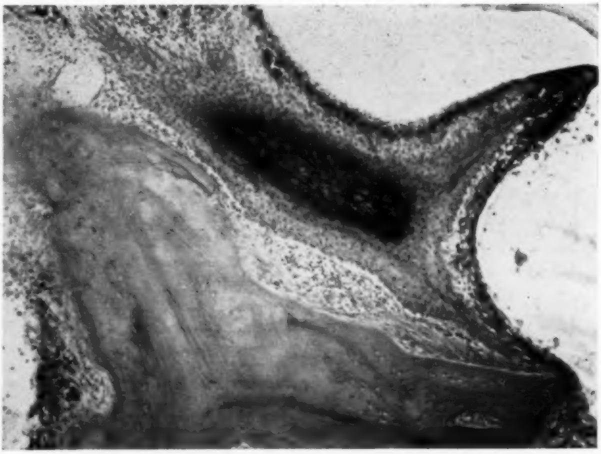

Fig. 7.—Higher magnification of Figure 6: A cholesteatomatous cone grows independently within the submucous layer of connective tissue.

In the case of a man 23 years of age who died 3 days after radical operation for otogenic meningitis and brain abscess due to Shrapnell cholesteatoma there is histological evidence of a small anterior superior marginal perforation in the right ear (Fig. 3). The cholesteatoma is about to invade the epitympanic recess which is filled with connective tissue. Behind the superior margin of the perforation the submucous connective tissue is directly covered by the matrix of the cholesteatoma. It is here that solid cones of basal cells of the Malpighian layer of the matrix proliferate into the connective tissue which shows inflammatory infiltrations. In Figure 4 a solid cone of basal cells of this kind can be recognized which infiltrates the connective tissue and is about to separate into cholesteatomatous cones. The behavior of the actively growing matrix towards the surrounding bony tissues and the reaction of this bone towards the growth of a cholesteatoma can be seen in the same case.

Figure 5 shows one large cone of a cholesteatoma in the center of the epitympanic connective tissue. Next to it, close to the bone there are several additional solid cones. Between the matrix and the bone a layer of connective tissue is regularly found. The matrix thus respects the border of the bone regularly and in all places. As the cholesteatoma continues to grow at the expense of the connective

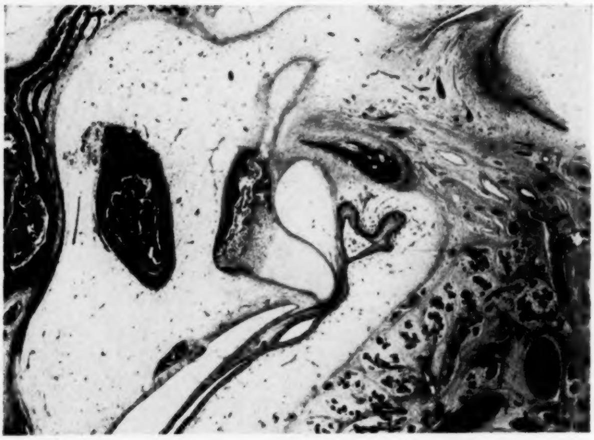

Fig. 8.—Vertical section of the middle ear of an embryo (6th month). Hyperkeratosis and acanthosis of the epidermis of the external auditory meatus adjoining the tympanic membrane anteriorly and superiorly.

tissue there is narrowing ossification of the connective tissue in the immediate neighborhood of the cholesteatoma cones.

On the other hand, how does the matrix of adolescent cases of cholesteatoma with large superior marginal perforation grow? From a group of eight histological specimens we choose the case of a 15 year old patient who died from an otogenic sepsis due to a cholesteatoma which had escaped diagnoses. In the horizontal section (Fig. 6) there are the almost complete defect of the tympanic membrane, the cholesteatoma in the tympanon and its infiltrating cones continuing to a considerable degree into the cells of the mastoid. Within the only partially pneumatized cells of good anlage of the periphery there are several solid cones of basal cells (Fig. 7). They grow independently, i.e., without any evidence of congestive pressure, within the submucous layer of connective tissue. The overlying intact epithelium of the mucous membrane is lifted off. Below, the basal cells continue to proliferate and finally separate into cholesteatomatous cones. The mucous membrane of the middle ear reacts in certain places with a loosening of its cuboidal epithelium and an inflammatory exudate discharged into the lumen of the cells. In other places the mucous membrane is invaded and perforated by the cholesteatoma

and it is now that squamae of the cholesteatoma are discharged into the lumen of the cells. The surrounding bone reacts in the manner already described.

The matrix of cholesteatomas associated with large defects of the tympanic membrane are therefore by no means harmless like an attempt at healing. To the contrary, the matrix proliferates within the layers of the submucous connective tissue as actively as the cones of a cholesteatoma of Shrapnell's membrane. The active growth of the matrix and the inflammatory reaction changing the structure of the bone occur at the onset of the disease and within the same connective tissue without mutual interference. Only when desquamated epithelium is accumulated in the widened cones of the cholesteatoma, and a congestion and infection of this material takes place, only then the secondarily sclerosed bone will undergo osteoclastic lysis. This further increase in size of the cholesteatoma at the expense of the surrounding bone only occurs at a late stage of the disease and is therefore typical of cases of long standing. A much more important factor for the comprehension of the entire pathogenesis is, however, the active proliferation of the matrix found in any type of early infantile cholesteatoma. This proliferative growth of the matrix occurs—and this is the second important observation—at the expense of the submucous connective tissue filling the remaining cells of the infant.

What is the origin of this special energy of growth typical of all cholesteatomas of the middle ear?

The answer to this question may also succeed in determining the exact location of the development of a cholesteatoma. It is known that as a rule cholesteatomas of the middle ear are associated with superior marginal perforations of the tympanic membrane. The supporters of the immigration theory will readily search for the origin of the special energy of growth in the epidermis of the external auditory meatus adjoining the tympanic membrane superiorly. In seven out of 21 cases of cholesteatoma Eigler has succeeded in demonstrating atypical growth in epidermis of the external auditory meatus which showed no signs of inflammation and was also associated with formation of epithelioid cysts.

In order to investigate this problem we have examined the epidermis of the external auditory meatus of embryos, newborns, and infants which had no ear pathology (53 cases, age: fifth fetal month to

Fig. 9.—Vertical section of the external auditory meatus and Shrapnell's membrane in an embryo (7½ month).

ten years). Our histological results were surprising: all healthy embryos, newborns, and infants show an increased growth in the sense of hyperkeratosis and acanthosis in a well defined section of the epidermis of the external auditory meatus adjoining the tympanic membrane anteriorly and superiorly (Fig. 8). Thickening of the stratum corneum of the epidermis of the external auditory meatus can be found in a section adjoining the tympanic membrane as early as the sixth fetal month (Fig. 9). In the eighth fetal month there is a limited hyperkeratosis and acanthosis of this section and it is also found regularly in all newborns (Fig. 10). In certain cases these changes in the stratum corneum resemble a cholesteatoma of the auditory meatus and a comparison to similar processes of cholesteatomas of the middle ear suggests itself.

At the time of birth and during the first months of life there is always a special tendency toward growth in the epidermis of the external auditory meatus adjoining the tympanic membrane superiorly. Therefore the assumption is evident that the special energy of growth so typical of the matrix of cholesteatoma may be derived from this section of epidermis and it seems probable that the cholesteatoma invading the middle ear starts from here either through a superior marginal perforation or—as we shall see—even without a primary

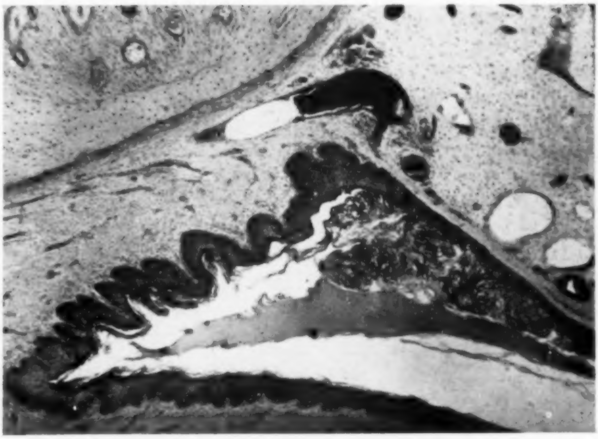

Fig. 10.—Vertical section of the external auditory meatus and Shrapnell's membrane in a newly born.

perforation. The findings in the matrix of the cholesteatoma and in the normal epidermis of the external auditory meatus therefore confirm Habermann's immigration theory.

Whether this immigration of epidermis is responsible for all types of cholesteatoma shall be investigated first in cases whose pathogenesis is known:

1) This includes the traumatic cholesteatoma. Only recently Escher reported an additional case of this type in which the epidermis adjoining the tympanic membrane was incarcerated in a longitudinal fracture of the petrous bone. After this dislodgement of the section of epidermis possessing special proliferative energy into the middle ear —almost representing an experiment—there developed a large cholesteatoma and since the well pneumatized mastoid contained no connective tissue the cholesteatoma expanded relative to its accumulation of congested epithelial cells at the expense of the surrounding bone.

2) The classical development of cholesteatoma during the course of an infantile necrotizing otitis media as first described by Habermann has been investigated conclusively also.

Fig. 11.—Otitis media necroticans in an 11 month old boy. Horizontal section. Nearly total marginal perforation of the tympanic membrane.

The case of an 11 month old boy who died of an osteomyelitis of the petrous bone may serve as an example (Fig. 11). The irritation of the necrotizing otitis media 20 days after the occurrence of a large marginal perforation of the tympanic membrane resulted in an active proliferation of the basal cells in the stratum germinativum of the adjoining epidermis of the external auditory meatus (Fig. 12). In the area of the superior marginal perforation the solid cones of basal cells partially invade the submucous connective tissue and partially invade the middle ear along the surface. The proliferation once started does not stop at the intact epithelium of the mucous membrane. This epithelium is now being undermined by the cones of the cholesteatoma, lifted off its connective tissue base, and displaced into the lumen.

3) Several years ago I reported two cases of another type of development concerning the rare cholesteatomas associated with small posterior superior marginal perforations: During the course of a subacute infantile mastoiditis the pus may perforate directly into the external auditory meatus posteriorly and superiorly in front of the tympanic membrane. The epidermis of the external meatus then grows through the fistula into the middle ear.

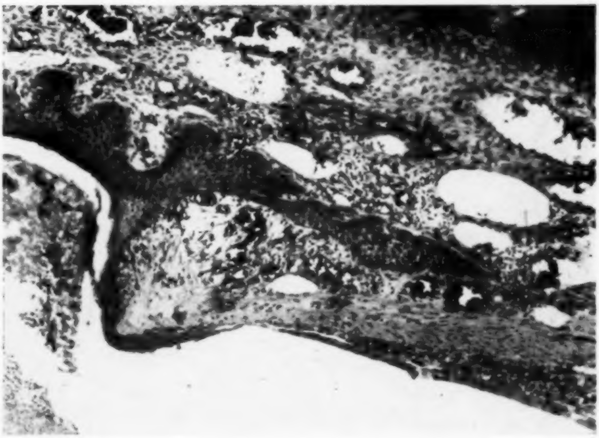

Fig. 12.—Higher magnification of Figure 11: In the area of the superior marginal perforation cones of basal cells partially invades the submucous connective tissue, partially invade the middle ear along the surface.

In these cases of cholesteatoma whose pathogenesis is known the matrix is unmistakably derived from the epidermis adjoining the tympanic membrane and possesses special proliferative energy. From here it invades the middle ear by way of a superior marginal perforation. Among the factors inducing growth the traumatic dislodgement represents an exception which is practically of no importance, whereas the irritation provided by a necrotizing or fistulizing otitis media contributes decisively in many cases.

We have now arrived at the last and most important question:

A necrotizing inflammation lacking, what are the other possibilities for symptomless development of a cholesteatoma of the middle ear from the epidermis of the external auditory meatus adjoining the tympanic membrane superiorly?

To answer this question we have examined 40 histological specimens of infantile cases of acute otitis media without perforations of the tympanic membrane.

In all of these cases more or less pronounced evidence of hyperkeratosis and acanthosis was found in that part of the epidermis of

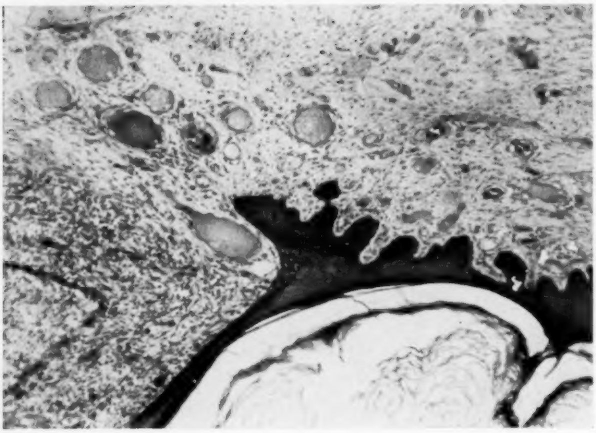

Fig. 13.—Vertical section through the external auditory meatus and Shrapnell's membrane in the case of an 11 month old girl suffering from an acute otitis media.

the external auditory meatus that adjoins the tympanic membrane superiorly. In some cases there are solid cones of cells derived from the layer of basal cells which invade the epitympanal area of the middle ear. The area of the epidermis of the external auditory meatus which already has special energy to grow during the first months of life therefore shows additional basal cell proliferation since this is stimulated in early infancy by the irritation provided by acute otitis media.

An 11 months old girl having died from Marfan's syndrome may serve as example (Fig. 13). The vertical section through the acutely inflamed left ear shows distinct hyperkeratosis and acanthosis with desquamation of horny lamellae in the epidermis of the external auditory meatus adjoining Shrapnell's membrane. Acanthosis is found in the layer of basal cells and there is a large solid cone of cells (Fig. 14) which grows actively, towards the connective tissue in Prussak's space which shows inflammatory changes and this cone may develop into a cholesteatoma.

These findings confirm the assumption that the latent energy of growth of the epidermis of the external auditory meatus can be activated by the irritation provided by acute inflammations of the

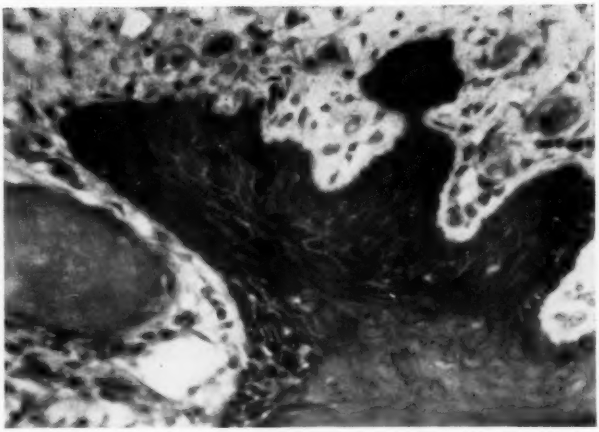

Fig. 14.—Higher magnification of Figure 13: A solid cone of basal cells grows actively toward the connective tissue in Prussak's space.

middle ear. Cholesteatoma can thus develop within an intact tympanic membrane since inflammations of the middle ear in early infancy demonstrate a special affinity to the already preformed cells of the infantile ear which only lack pneumatization in accordance with Lange, Albrecht, Steurer, and Saxén. This occurs as acanthotic cones of basal cells, which are derived from the epidermis adjoining the tympanic membrane and from Shrapnell's membrane respectively, proliferate into the connective tissue still present in the epitympanon. Here they separate due to hyperkeratosis and form an initially well defined cholesteatoma. Due to the continuing proliferation this will eventually perforate from the epitympanon into the external auditory meatus by way of a small superior marginal perforation.

It is unfortunate that we do not yet have histological specimens of acute otitis media in which proliferated cones of basal cells of the auditory epidermis have penetrated the connective tissue remaining in the epitympanon and forming a cholesteatoma. Histological proof of an inflammatory development of a cholesteatoma within an intact Shrapnell's membrane is therefore still lacking. Clinical observation of a cholesteatoma behind an intact tympanic membrane must therefore serve as provisional substitute. This is the rare possibility so often quoted as a main argument by supporters of the metaplasia of mucous membrane theory. A case of this type may therefore well

serve as the crucial experiment for investigating the pathogenesis of cholesteatoma.

It is the case of a little boy, 6½ years of age, who since the age of 5 suffered from a suppuration of the middle ear that was at times fetid. At this time the attending specialist found a perforation of Shrapnell's membrane and several cholesteatomatous nodules and therefore recommended radical operation. Only a short time later the discharge from the ear ceased spontaneously and the perforation of Shrapnell's membrane healed, leaving a small scar. Only a moderate conduction deafness and a shaded mastoid as seen in the x-ray film remained. Despite this spontaneous healing the boy was operated on under the tentative diagnosis of cholesteatoma behind an intact tympanic membrane. The operative findings revealed a mastoid with extensive cells containing plenty of granulation tissue. There was a cholesteatoma in the antrum running from the aditus along the tegmen and towards the angle of sinus and dura. Behind the *intact* membrane of Shrapnell there was an additional nodule of the cholesteatoma, approximately the size of a bean that originated from the inner surface of Shrapnell's membrane and developed towards the tympanum proper and tube. While carefully removing the cholesteatoma its origin at the inner surface of Shrapnell's membrane was distinctly visualized with the oto-microscope. The narrow stem of the matrix rooting in Shrapnell's membrane caused a rupture of this membrane when severance was attempted. In this case the cholesteatoma has developed primarily from the initially intact membrane of Shrapnell.

On the basis of this history the assumption may be made with the greatest probabily that in this case a cholesteatoma developed primarily from the initially intact membrane of Shrapnell in the manner we have described above. Eventually the congested epithelial masses perforated the weakened point of origin and infected material of the cholesteatoma was during some time discharged into the external auditory meatus through the perforation in Shrapnell's membrane. The congested masses having been discharged and the inflammatory reaction having subsided the perforation in Shrapnell's membrane closed spontaneously but the cholesteatoma continued to grow behind the now healed tympanic membrane.

This clinical observation of the development of a cholesteatoma behind the intact tympanic membrane in our opinion completes the histological findings according to which cholesteatomas of Shrapnell's

membrane are caused by an inflammatory immigration of epidermis of the external auditory meatus.

On the basis of clinical observations and especially of histological investigations of infant petrous bones we arrive therefore at the following opinion concerning the pathogenesis of the cholesteatoma of the middle ear.

1) As a rule all types of cholesteatoma of the middle ear develop by immigration of stratified squamous epithelium from the epidermis of the external auditory meatus or Shrapnell's membrane.

2) On one hand this immigration is made possible by the normally inborn energy of growth which is present in early infancy in the epidermis of the auditory canal adjoining the tympanic membrane superiorly. On the other hand active growth of the cones of basal cells is enhanced by submucous connective tissue filling the incompletely pneumatized epitympanal spaces and cells.

3) The immigration of stratified squamous epithelium is as a rule caused by inflammation of the middle ear. In early infancy the necrotizing otitis media causes frequently large superior marginal perforations of the tympanic membrane. Exceptionally a small posterior marginal perforation is due to an empyema of the mastoid that has perforated into the external auditory meatus. More often the immigration of the squamous epithelium is due to acute inflammations of the middle ear of infants since they initiate a proliferation of basal cells within the epidermis adjoining the tympanic membrane or within the intact membrane of Shrapnell. Upon this the cholesteatoma initially grows behind the intact tympanic membrane and perforates only secondarily through a small superior marginal perforation into the external auditory meatus. Only when desquamated epithelium is accumulated and is subject of infection, the pressure of the cholesteatomatous material will produce an osteoclastic destruction of the adjoining bone.

4) The chronic irritation provided by the cholesteatomatous suppuration of the middle ear causes an arrest of pneumatization of the initially cell-containing mastoid, and a secondary sclerosis of the bone. Only when desquamated epithelium is accumulated and is subject to infection, the pressure of the cholesteatomatous material will produce destruction of the adjoining bone.

The *therapy* of cholesteatoma depends as stated to a large extent on the opinions concerning its pathogenesis. Tumarkin, a supporter

of the metaplasis theory, recommends the decompression of the masses of the cholesteatoma by an operation that opens the aditus and antrum via the external auditory meatus. After establishing a large approach to the cavity of the cholesteatoma the disease is said to heal spontaneously. Tumarkin even says: "A complete restitutio ad integrum back to the normal pavement epithelium condition takes place." To our knowledge this fortunate regression of the matrix has never been proven histologically. But even the founders of the immigration theory, Bezold, Manasse, et al., regard the immigration of epidermis as an attempt at healing and therefore recommend only a wide opening and decompression of the cavity of the cholesteatoma. Since then, not only the degree of radical operation and behavior towards the matrix are being discussed but even the indication for surgical treatment of the cholesteatoma per se.

On the basis of the opinion on the pathogenesis of cholesteatomas developed in this paper we adhere to the following therapeutic principles:

1) All forms of cholesteatoma should be treated surgically and as early as possible.

2) In all cases of cholesteatoma the complete removal of the matrix, of all cell systems, and all connective tissue contained therein is necessary.

3) Maintenance or improvement of relatively well preserved auditory capacity must be achieved by carefully avoiding injury to the auditory ossicles when removing a cholesteatoma and by closure of a perforated tympanic membrane in which plastic surgery is employed.

4) In extensive loss of auditory capacity due to lesions in the sound conductive apparatus functional improvement should be the ultimate goal. With the help of plastic surgery a new sound conductive system has to be built.

5) To prevent development of a cholesteatoma a preventive antro-atticotomy and removal of connective tissue layers in the epitympanum is recommended in cases of recurrent infantile acute otitis media.

Ramistrasse, 100

BIBLIOGRAPHY

Albrecht, W.: Mittelohreiterung und Pneumatisation des Warzenfortsatzes. Z. Hals- usw. Heilk. 10:55, 1924; Zur Frage der Pneumatisation des Mittelohrs. Histologische Untersuchungen am Schläfenbein des Neugeborenen. Acta oto-laryng. Stockh. 14:221, 1930; Zur Cholesteatomgenese. Z. Hals- usw. Heilk. 32:517, 1933.

Bezold, F.: Ueber das Cholesteatom des Mittelohrs. Z. Ohrenheilk. 21:252, 1891.

Diamant, Marcus: Chronic Otitis. Karger, Basel, 1952.

Eigler, G.: Das Verhalten der hinteren oberen Gehörgangsepidermis bei randständigen Perforationen (Cholesteatomeiterungen). Hals-Nas.-Ohrenarzt. 2:281, 1951.

Escher, Franz: Traumatische Cholesteatome. Pract. oto-rhino-laryng. Basel, 16:32, 1954.

Habermann, G.: Zur Entstehung des Cholesteatoms des Mittelohrs. Arch. Ohrenheilk. 27:42, 1889.

Lange, W.: Ueber die Mittelohr-Cholesteatome. Z. Hals-usw. Heilk. 11:250, 1925.

Link, R.: Das Cholesteatom des Schläfenbeins. Bergmann, Wiesbaden, 1914; Zur Morphogenese des knöchernen Gehörganges (ihre Bedeutung für die Entstehung des Cholesteatoms). Zbl. Hals-Nas.-Ohrenheilk. 44:7, 1952.

Manasse, P.: Handb. Path. Anat. menschl. Ohr. Bergmann, Wiesbaden, 1917.

Marx, H.: Kurzes Handb. Ohrenheilk. Fischer, Jena, 1938.

Mündnich, K.: Ueber das Hamartom im Bereich der Paukenhöhle. Mschr. Ohrenheilk. 73:239, 1939.

Rüedi, L.: Zur Klinik und Therapie des Mittelohrcholesteatoms. Schweiz. Med. Wschr. 19:411, 1934; Die Mittelohrraumentwicklung vom 5. Embryonalmonat bis zum 10. Lebensjahr. Acta oto-laryng. Stockh., Suppl. 22, 1937; Mittelohrraumentwicklung und Mittelohrentzündung. Z. Hals- usw. Heilk. 45:13, 1939.

Saxén, A., und Ojala, L.: Pathogenesis of Middle-Ear Cholesteatoma Arising from Shrapnell's Membrane (Attic Cholesteatoma). Acta oto-laryng. Stockh. Suppl. 100, 33, 1952.

Schwarz, M.: Die Schleimhäute des Ohres und der Luftwege. Springer, Berlin, 1949; Die Einsenkung der Shrapnell'schen Membran und ihre klinische Bedeutung. Arch. Ohr-Nas.-Kehlkopfheilk. 131:16, 1932.

Steurer, O.: Zur Pathogenese der Mittelohrcholesteatome. Zbl. Hals-Nas.-Ohrenheilk. 14:234, 1930.

Teed, R. W.: Cholesteatoma Verum Tympani: Its Relationship to the First Epibranchial Placode. Arch. of Otolaryng. 24:455, 1936.

Tumarkin, J. A.: A Contribution to the Study of Middle-Ear Suppuration with Special Reference to the Pathogeny and Treatment of Cholesteatoma. J. Laryng. and Otol. 53:685, 737, 1938; Attic Suppuration. J. Laryng. and Otol. 64:611, 1950; Discussion on the Management of Chronic Otitis Media. Proc. R. Soc. Med. 46:379, 1953.

Wittmaack, K.: Ueber die normale und die pathologische Pneumatisation des Schläfenbeins. Fischer, Jena, 1918.

XXII

THE CHOANAL POLYP

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The subject of the mucous polyp of the nose and sinuses has interested investigators for many years. The benign inflammatory or allergic polyp is unquestionably the most frequently encountered new growth in the nasal fossa and paranasal sinuses. The choanal polyp occurs with much less frequency and, although it is grossly and microscopically similar to the ordinary nasal polyp, it exhibits certain characteristics that make it distinctive and, as a result, is the subject of many articles and discussions. The purpose of this paper, therefore, is to determine, if possible, whether any additional information may be obtained on this subject by a review of the literature and a study of a number of case records.

In 1691 the great Dutch anatomist, Fredrik Ruysch,[37] published a book entitled *One Hundred Remarks on Surgery and Anatomy* in which he stated: "Barbers and even the half-blind* are familiar with polyps, in whatever state they may be; they are not familiar, however, with the base from which the polyp erupts. There are those who think a polyp obstructs only the nose; others assert that it may occupy the throat and even the ears. On two occasions we have found polyps in the hollow of the fourth pair of bones (others call them the third pair) of the jaw, named by Highmore, the antrum."

One of the earliest descriptions of the choanal polyp appeared about 60 years after Ruysch. In 1753, Palfyn[35] wrote a description of a polyp in the nasopharynx which he observed in a girl and, because

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* Translator's note: Ruysch apparently exhibits here some professional pride to the detriment of the barber-surgeons of the 17th century, mentioning them in the same breath with "the bleary-eyed" or "the half-blind."

I was called to examine a girl whose interior nasal membrane had the account is so graphic, it is worth quoting in part: "Some years ago been so greatly stretched by a mass of humors that it formed a tumor as large as a pigeon's egg hanging behind the uvula. This excrescence was of the type generally known as polyp; the tumor was round, smooth, whitish, insensitive, soft, and consequently of the type which is curable. The patient had difficulty speaking and was able to breathe only with her mouth open."

In the 19th century a number of interesting observations were published, and apparently more attention was given to this odd type of nasal growth. Semeleder (1866)[39] described a number of patients who had what he termed a nasopharyngeal polyp, and he believed that it originated on the superior border of the soft and hard palate. A sketch of a polypoid growth in the nasopharynx appeared in his book. Kubo[21] mentioned that Scheck (1884) also had a drawing of what was probably a choanal polyp, and he stated that the origin of the polyp was difficult to determine. According to Killian,[20] Moldenhauer (1886) emphasized that the choanal polyp was a special type and thought that it originated in the posterior end of the middle turbinate.

In 1887, Sands[38] reported the removal of a nasopharyngeal polyp by means of a galvanic ecraseur, and he considered the probable origin of this particular one "from in front of the foramen lacerum medium." He also stated that most polyps of this type were attached to the skull by a broad base. In the same year, Lange[26] described a hook that he had devised for the removal of choanal polyps and he expressed the opinion that the middle turbinate was the favorite site of origin of nasal polyps, especially the choanal polyp.

Although no specific reference was made to a choanal polyp, Zuckerkandl (1891)[50] in his book had an illustration of a pedicle originating in an antrum and passing into the nose through a large accessory ostium (Fig. 1). In another drawing in the same book, a polyp is shown arising in the sphenoid sinus and extending by a long thin pedicle through the sphenoid ostium and into the posterior nasal area (Fig. 2).

DESCRIPTION OF ORIGIN

The mucous polyp protruding from a choana and appearing in the nasopharynx has been designated by a variety of names, viz.,

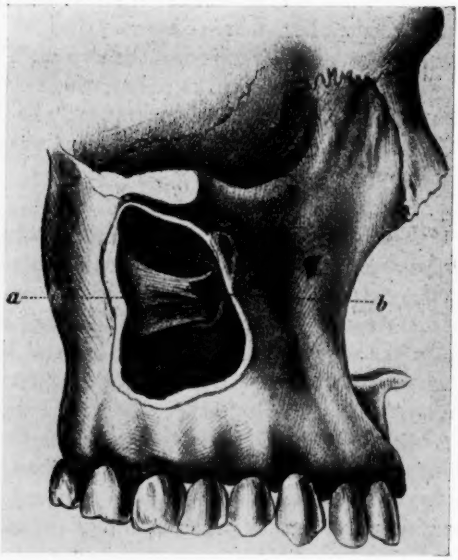

Fig. 1.—Taken from Zuckerkandl.

nasoantral polyp, benign nasopharyngeal polyp, postnasal polyp, antrochoanal polyp, recurring polyp of youth, solitary choanal polyp, retronasal polyp, and Killian polyp.[14,23,27,45] This type of mucous polyp is not histologically different from the ordinary nasal polyp but is dissimilar in origin, entrance to the nasal cavity, and location in the nasal tract.

In 1906, Killian[20] presented a paper on the origin of the choanal polyp and, from a detailed study of several cases, expressed the opinion that it arose from the maxillary sinus and gained access to the nasal cavity through the accessory ostium. He stated that he had been able to trace the pedicle from a choanal polyp into the middle meatus of the nose and thence forward to the accessory ostium where the stalk entered the antrum. By using a probe with a right angle tip and passing it around the pedicle, he was able to show that there was no attachment to the margins of the accessory ostium. Although he was able to demonstrate this repeatedly, in no instance did he inspect the interior of the maxillary sinus.

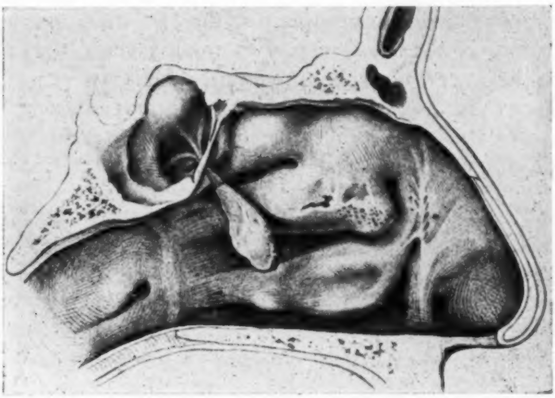

Fig. 2.—Taken from Zuckerkandl.

A total of 22 cases was recorded, and in each instance the polyp was removed by avulsion and without cutting the pedicle. By such a procedure he was able to obtain pedicles which were almost as long as the attached polyps. After the removal, a quantity of serosanguineous fluid usually escaped from the patient's nose. He surmised, therefore, that there was an intramaxillary stalk which was distended with cystic fluid. During the process of extraction this stalk ruptured, leaving a long thin collapsed cyst wall.

FINDINGS WITHIN THE SINUS

After Killian[20] showed that a pedicle passed through the accessory ostium, the origin of the pedicle within the maxillary sinus was sought. In 1909, Brown Kelly[19] reported 15 cases of nasoantral polyps and in 11 of these he opened the antrum by the Caldwell-Luc procedure and demonstrated a connection between the lining membrane of the sinus and the polyp. In this series he also noted that a cyst was often present within the antrum, but added that a polyp or thickened membrane might be present instead of a cyst. Thus, in seven instances he found that a large cyst occupied the antrum cavity; in 4 others there was no cyst but the mucosa was polypoid in two and it was thickened in two. In all instances there was a large accessory ostium providing ready communication between the antrum and nasal fossa.

Brown Kelly[18] previously made some interesting observations when post mortem he opened 200 maxillary sinuses. In 21 sinuses he found accessory openings, and in five of the 21 the lining mucosa was cystic. In four of the five sinuses with cystic mucosa, the cysts were situated on the inner wall of the antrum near the accessory ostium. As a result of these findings, he stated that "the comparatively frequent association of cysts to the lining membrane with accessory openings would seem to indicate a relation between the two conditions."

In 1909, Ino Kubo,[21] a student of Killian, returned to Japan and, in order to learn more of the origin of the polyp, opened the maxillary sinuses on three patients. He observed stalks passing from the lateral walls of the sinuses to the accessory ostia. In 1913, Kubo[22] reported an addition.l 36 cases. In this series he found that the antrum was the site of origin in 27; three arose from the sphenoid and six were of unknown origin. Bilateral polyps were found in two cases. He noted also that in 19 of the 26 he opened he found polyps or cysts in the antrum. Since then numerous observers have reported similar findings.

About the same time that Kubo made his report, K. Lang[24,25] expressed an opposite opinion. He stated that the choanal polyp seldom originated in the antrum and based this opinion on his examinations with the antroscope. He made an opening through the canine fossa in several instances and introduced an antroscope. In none of the patients examined in this manner could he see a polyp or stalk within the antrum.

ROOT OF THE PEDICLE

Only rarely has any location other than the maxillary sinus been reported as the site of origin of the choanal polyp. There have been a number of sphenochoanal polyps reported, and some choanal polyps from the ethmoid cells have been described.[43] McKenzie[28] stated that polyps of this type did not come from the ethmoidal region since polyps growing there are not constricted. He theorized that if they were constricted at their origin, then the cell walls would be destroyed by the growth of the tumor. This would not take place in the sphenoid or maxillary sinuses, he thought, because the bone around the ostia of these sinuses was too dense and too hard to be disrupted by the polyp.

The recurrence of a postnasal polyp originating in the left sphenoid sinus was reported by Moore.[33] The origin of the pedicle

was located at the lower margin of the sphenoid ostium. He stated that until then (1918) only nine cases of sphenochoanal polypi had been reported in the literature, and all of them were in adults between 20 and 30 years of age. Kubo,[22] Syme,[43] and Ewing[10] were others who reported sphenochoanal polyps.

It has been already mentioned that Killian[20] stated that the choanal polyp came from the antrum, but he did not open the sinus and inspect the interior. Brown Kelly[19] went a step further and opened several maxillary sinuses and showed that the choanal polyp originated in them but made no mention of the site of origin within the sinus. Although he apparently preceded Kubo in opening the antrum and demonstrating the origin of the polyp, Kubo[22] was the first to search for the exact site of origin and report his findings.

Kubo[22] noted the following sites within the maxillary sinus:

Posterior wall	10 times
Inferior wall	8 times
Medial wall	7 times
Lateral wall	6 times
Superior wall	3 times
Anterior wall	1 time

In two cases the site was not determined.

Forschner[11] reported finding the pedicle arising from the floor and medial wall of the antrum, and Wishart[49] found the origin to be usually on the posterior or outer wall, but in no instance was there any attachment to the margins of the ostium. Syme[43] and Mithoefer[32] recorded the region of the maxillary ostium as the site of origin, while Ewing[10] found the site to be on the inner antral wall close to the accessory ostium in most instances. Baum,[3] Shea,[41] Dawson,[8] and Davis[7] also described the site to be near the margin of the ostium. Stawraski[42] stated that he observed six pedicles coming from the upper lateral wall (recessus zygomaticus). A number of authors have reported that, except for the area of origin of the stalk, the remainder of the lining mucosa of the sinus may appear normal, but there has not been much evidence to back this assertion.[14,32,43]

Recently, Semenov[40] stated that the origin of the antrochoanal polyp was on the lateral wall of the maxillary sinus but commented

around the ostium the greatest irritation and discharge occurs. He thought that the explanation for the lateral wall being the site might lie in the embryology and histology of the maxillary sinus. He stated that the mucosa lining of the lateral portion was extremely thin and delicate and, because it has a long line of lymphatic drainage, it is subject to much edema.

Ash and Raum[1] were in essential agreement with this idea for they stated that the choanal polyp usually arose from the lateral wall of the antrum since the mucosa was very thin and rudimentary and the blood supply and lymphatic drainage were more vulnerable.

MECHANISM OF DEVELOPMENT

Since Killian's observation in 1906, there has been considerable speculation about the conversion of a simple antral polyp or of edema-
that one would expect a point nearer to the ostium to be the site since
tous antral mucosa into a choanal polyp. Killian believed that the choanal polyp arose from a simple antral polyp which found its way to a wide accessory ostium and passed through it into the nasal chamber. He suggested that blowing the nose or irrigation and inflation of the maxillary sinus could force a polyp from the sinus into the nose. After the polyp became fixed in the ostium, congestion and growth followed, and it moved posteriorly to the roomy nasopharynx, where further enlargement occurred. The result, then, was an hour-glass sac with the constriction at the ostium, one portion being in the antrum and the other part in the nose or nasopharynx.[20]

Von Bajkay[2] observed 11 choanal polyps which originated in the maxillary sinus and repeated Killian's idea that the polyps could get into the middle meatus by blowing the nose or by syringing the maxillary sinus.

Brown Kelly[19] was of the opinion that when a cyst, polyp, or thickened lining membrane was present within a maxillary sinus and was accompanied by a large accessory ostium or natural ostium, a choanal polyp could develop. He offered no theory, however, as to how this took place. Syme[43] also thought that a polyp arose within the antrum in the neighborhood of the maxillary ostium and in some manner found its way through it into the middle meatus. The passage to the choana was then dependent on the configuration of the meatus, the air current, and the effect of gravity. He believed, in addition, that a similar process could take place in any of the accessory sinuses,

especially the posterior group and reported that he had demonstrated a sphenochoanal polyp which took origin on the inner lip of the edge of the sphenoid ostium.

Baum[3] and Dingley[9] had somewhat similar ideas. Baum concluded from his observations that inflamed mucosa within the antrum was forced by the inflammatory pressure into the middle meatus. In addition to this conception, Dingley also thought that a choanal polyp could be the result of a cystic herniation which was forced out of the antrum by increasing pressure.

Citelli,[4] Hirsch,[15,16] and Forschner[11] considered the choanal polyp to be an incarcerated prolapse of swollen mucosa which arose from the floor or medial wall of the antrum and pressed into the accessory or maxillary ostium. Hirsch postulated that the incarcerated portion continued to enlarge as a result of passive congestion and inflammation but that a pedicle was formed within the antrum as the edema subsided and the mucosa shrank. However, the insertion site of the pedicle within the antrum remained in a state of chronic inflammation and edema.

Stawraski[42] also held that the choanal polyp formed as a result of edematous mucous membrane prolapsing through a large accessory ostium and, in addition, stated that a retronasal polyp could not form unless a widened ostium was present.

The idea of a cyst filling the antrum and pushing into the nose was mentioned by Ewing,[10] but he added that this theory was not acceptable to him. Neither did he accept Killian's idea that the polyp entered the middle meatus from the antrum as a result of blowing the nose. He believed, however, that during the act of hawking or sniffing it was possible for a bit of edematous antral mucosa or an antral polyp to be sucked through an accessory ostium into the middle meatus. He thought that sniffing would produce negative pressure in the nasal passage and that air passing from the antrum through the accessory ostium would carry a polyp near this opening into the nasal cavity.

Ewing[10] also suggested that the formation of the choanal polyp was the result of a developmental error. He recalled that the space formed by absorption of the cancellous bone in the body of the superior maxilla is lined by a hollow bud of epithelium that extends outward from the middle meatus. He thought it possible for a small

fold of the mucosa to form in the region of the natural or accessory ostium, or between the two ostia. This epithelial fold then could possibly be sucked through an ostium into the middle meatus during sniffing or hawking and develop into an antrochoanal polyp.

Proetz[36] states that it would be unlikely for a small antrum polyp to be forced into the middle meatus either by blowing or sniffing. Blowing the nose forces air into the antrum and while it is theoretically possible that a very small polyp lurking in the sinus just at the ostium might be popped out on the rebound, this is unlikely. In the first place, the pressure change within the nasal chamber is so slight that it is doubtful that enough negative pressure could be created; and in the second place, the polyp within the antrum would have to be displaced by an equal amount of air. If such a physical change could be accomplished by sniffing, then only a minute antral polyp at or very close to the edge of the accessory ostium could be replaced by an equal volume of air since the pressure changes are so small. If there are two openings into the sinus then a polyp at the meatus will be under equal pressure, from within and without, at all times and no extrusion can take place. Proetz believes that a markedly edematous sinus membrane, the edema resulting from any cause, or increased air pressure in the sinus caused by it in case the ostium were blocked, could conceivably push a small antral polyp through a flat natural or accessory ostium. This polyp could then become incarcerated and increase in size as others have suggested.

The movement of a polyp from sinus to nose would take place more likely at an accessory ostium because, as Meyerson[30] has shown, the maxillary ostium frequently has depth. He demonstrated that the passage from the antrum to the nose may be tubular, S-shaped, funnel-shaped, straight, obliquely vertical, obliquely horizontal, or horizontal.

PATHOLOGY

There is general agreement among pathologists that choanal polyps do not differ histologically from the simple mucous polyp of the nose. Ash and Raum[1] stated that the number of mucous glands present in nasal polyps will vary with the site of origin, and they have asserted that polyps arising from the antrum may contain a few, especially if the origin was near the ostium. They also observed that polyps arising from the maxillary sinus and turbinates may show a

greater gland content than would be expected in a section of the original mucosa.

Semenov[40] concurred with the idea that when polyps arose from membrane rich in glands these glandular elements appeared in the polyp. He stated also that the choanal polyp had an edematous stroma with round cell infiltration but usually no eosinophilic cells.

Heck, Hallberg, and Williams[14] noted that a choanal polyp was less likely to show eosinophilic infiltration than a nasal polyp. They made a detailed analysis and comparison of 64 cases of antrochoanal polyp with an equal number of nasal polyp cases, and concluded that both grossly and microscopically there were very few differences between the two types.

Walsh and Lindsay[48] believed that nasal polyps could be divided into two groups; those infiltrated with numerous eosinophils and those with few or no eosinophils. After reviewing many cases and correlating the clinical and histological findings, it was their conclusion that the polyps without eosinophilic infiltration were invariably associated with infection.

Hansel[13] felt that a distinction should be made between the nasal polyp of true allergic origin, the polyp of apparent allergic origin, whether it occurred with or without secondary infection, and those polyps which resulted secondarily from a suppurative sinusitis. He excluded the true choanal polyp, mulberry hypertrophies, and papillary hypertrophies from all other nasal polyps.

FREQUENCY OF OCCURRENCE

The frequency of occurrence of the choanal polyp has not been ascertained with any degree of accuracy because each author has used a different method of computation. A brief summary of some of the reports, therefore, should give an indication of the relative infrequency of this condition.

Syme[44] noted the frequency of occurrence of the choanal polyp among patients with maxillary sinus disease. He reported that the radical operation was performed 393 times among 878 cases of antral disease. Choanal polyps were found in 26 instances (6.6 per cent) and in two of these 26 there was a choanal polyp on both sides.

By way of comparison, Morwitz[34] stated that the choanal polyp occurs in about three per cent of patients with antral disease. Mit-

hoefer[32] found a much higher incidence but limited his analysis of cases to those requiring sinus surgery. He reported 48 radical maxillary operations for the removal of hyperplasia not associated with suppuration, and noted that solitary nasal and retronasal polyps were present six times (12.5 per cent).

In 1918, Guthrie,[12] in a paper on choanal polyps in children, stated that 116 instances in patients under the age of 16 had been recorded in the literature and only 14 of these were in children under the age of ten.

Forschner[11] quoted Beco as stating that in 30,000 cases of nasal polyps he found only four cases in children, but of these, three had choanal polyps.

In a three year period, during which over 25,000 otorhinologic examinations were made, Meyers[29] found three patients each with a choanal polyp.

The statistics of Heck, Hallberg, and Williams[14] are perhaps the most significant. They reported 64 cases of choanal polyps, or 3.7 per cent, in 1720 patients who were found to have nasal polyps. The latter figure included all patients diagnosed as having nasal polyps during a ten year period. This comparison was most expressive because these authors were the only ones to specify the numerical relationship of choanal polyps to all nasal polyps.

DIAGNOSIS

The choanal polyp can usually be diagnosed by inspection of the nasal chamber and nasopharynx. It is typically round, smooth, gray or bluish in color, and may be seen on posterior rhinoscopy either projecting from a choana or completely filling the nasopharynx.[9,41,45,46] It is not attached to the edges of the choana or to the walls of the nasopharynx.[6,7] At times it may enlarge and drop below the level of the soft palate into the oropharynx.[8,22] A careful inspection will generally reveal a pedicle which, in most instances, will pass from the polyp anteriorly into the middle meatus. There may be a mucous secretion, purulent at times, either in the meatus in the vicinity of the pedicle or on the surface of the polyp. As a rule, it is not difficult to differentiate it from a nasopharyngeal fibroma or from other neoplasms peculiar to the nasopharynx.

Assistance in diagnosis may be obtained by roentgen study of the sinuses. Most authors agree that roentgenograms will frequently

demonstrate a cloudy or opaque antrum on the side of the polyp. However, evidence of involvement of sinuses on both sides is not uncommon.[14] Transillumination of the sinuses as an aid in diagnosis has been advocated by some, but others have felt that transillumination of the maxillary sinuses is of minor value and may, in some instances, give misleading information.[9,17,43]

TREATMENT

The most commonly recommended method for removal of the antrochoanal polyp and its pedicle has been the Caldwell-Luc operation. It is the opinion of most observers that the membrane from which the pedicle arises must be removed if recurrence of the polyp is to be prevented. This can be accomplished best by the external approach.

Von Bajkay[2] advised locating the origin of the choanal polyp and then performing a radical operation on whichever sinus was involved. He reported removing three polyps by opening the ethmoid cells but he did not describe the method he employed.

The hook, knife, scissors, and snare have also been used to sever the pedicle.[2,26,45,47] In addition to the likelihood of recurrence, the danger of bleeding from a cut or torn pedicle has been given as a reason against removal by one of these procedures.[22,32] However, this is a rare complication.

The use of radium has been reported as unsuccessful in the treatment of the choanal polyp.[2]

In as much as many polyps do not recur, a few authors have stated that a simple removal should be done unless one is dealing with a recurrence.[12,42,45]

REPORT OF CASES

The following cases are described briefly because of certain unusual features. There can be no doubt that each one had a choanal polyp. One apparently did not arise in the maxillary sinus and the presence of infection seemed to alter the clinical picture in the others.

CASE 1. Only one patient in this series had physical findings and roentgen studies which indicated that the polyp did not arise in the maxillary sinus. A 17 year old girl had right nasal obstruction and bleeding from the right nostril for three and one-half months. On examination, a gray mass of tissue was found presenting anteriorly in the right nasal fossa and a large grayish mass covered with

mucopus also filled the nasopharynx. The roentgenogram showed a soft tissue mass overlying the paranasal sinus area and the upper nasopharyngeal shadow. Detail, especially in the ethmoid area, was obscured.

The mass was removed by a right external ethmoidectomy and it was reported as being "choanal in origin." All the ethmoid cells were lined with thickened polypoid membrane. There was no recurrence of the mass after six years.

The pathologic report was chronic inflammation of the mucous membrane and a polyp with degenerative changes.

Case 2. A 49 year old male had pain through the left side of his face for a year, and left nasal obstruction for several years. A large polypoid mass, covered with mucopus, filled the left nasal fossa. The roentgenogram showed an opaque left antrum and haziness of the left frontal sinus and ethmoid cells. A large rounded tumefaction was noted in the right antrum. He could not be admitted to the hospital immediately so the polypoid mass was removed from the left nasal cavity and a month later he entered for sinus surgery.

A bilateral Caldwell-Luc operation was performed and thickened polypoid membrane, several polyps, and mucopus was removed from each antrum. He continued to complain of pain and nasal obstruction and a month later a large choanal polyp was noted on the left side but no note was made indicating the possible origin. The polyp was removed by snare and avulsion.

Microscopic examination of the polyp showed the surface epithelium to be pseudostratified columnar. The underlying connective tissue was markedly edematous and infiltrated with lymphocytes and plasma cells. In some areas there were moderate numbers of eosinophils.

Because of clinical evidence of continued suppuration, he was again admitted to the hospital 3 months later, at which time a left intranasal ethmoidectomy was performed.

There was no recurrence of the choanal polyp three years later.

Case 3. A 9 year old girl was first admitted to the hospital in November 1954 because of nasal obstruction and discharge. There was a negative history for allergy. There were polyps in the left nasal cavity and also a left choanal polyp. At surgery, the left antrum was irrigated but no mucopus was obtained. Several polyps were removed from the middle and superior meatuses using a snare. The choanal polyp was grasped with forceps and a thin pedicle which seemed to arise from the region of the middle turbinates was cut with a scissors.

The microscopic study of this tissue showed a polypoid structure containing a loose fibrillary stroma having a number of dilated lymphatic channels and proliferating capillaries. The border was pseudostratified columnar epithelium. There was considerable inflammatory reaction in the sections. The final diagnosis was nasal polyps.

She was readmitted in March 1955 and stated that nasal obstruction recurred shortly after the previous operation. Both sides of the nose were filled with mucopus and the left nasal fossa was blocked with a grayish white mass. At operation a large left choanal polyp was again found and removed. A large necrotic mass was also excised from the upper half of the left nasal cavity and an intranasal antrotomy was performed. Microscopic examination of the tissue showed it to be similar to that previously removed.

At this admission the differential blood count showed a blood eosinophilia of 3 per cent. A month later the differential was repeated and it then revealed 17.5 per cent eosinophils.

In February 1956 she was admitted for the third time because a large reddish polypoid mass protruded from the left nostril. At surgery several polyps were removed from the left side of the nose and polyps were also found within the antrum, high in the nasal chamber, and posteriorly hanging into the nasopharynx. There was no pathological report on this tissue.

The last admission was in May 1956 at which time she complained of left nasal obstruction. The left nasal cavity was completely filled with a polypoid mass and mucopus was noted on the surface. A left Caldwell-Luc operation was performed and edematous polypoid tissue was found filling the antrum, and a few polyps were noted in the left middle meatus. The pathological diagnosis again was nasal polyps and chronic inflammatory reaction.

RESULTS OF STUDY AND COMMENT

Because there has been no specific diagnostic number for the choanal polyp in the *Standard Nomenclature of Diseases and Operations*, it has been difficult to locate records of patients with this condition. The 31 cases studied for this report were located in the 38,185 pathological diagnoses recorded in the ear, nose, and throat pathology section at the medical center in the past 25 years, but probably do not represent the total number seen at the institution during that period.

TABLE I

AGE GROUPS OF 31 PATIENTS WITH CHOANAL POLYPS

Below 10 years of age	1
10 to 19	12
20 to 29	8
30 to 39	4
40 to 49	4
Over 50 years of age	2

Sex and Age Incidence. There were 21 females and ten males in this series. Most of the females were in the younger age groups: one was nine years old; ten were between ten and 19 years, inclusive; six were between 20 and 29 years; and four were in the 30 to 39 year group.

There were only two males under 20 years of age: two were between 20 and 29 years; four between 40 and 49 years; and two were over 50.

The ages of both males and females grouped together are summarized in Table I.

These figures agree with those submitted by Heck, Hallberg, and Williams, who noted that the choanal polyp occurred mostly in adults.

The sex ratio of the two series, however, was exactly opposite.

Roentgen Examination. The best series of cases with roentgen examination was reported by Heck, Hallberg, and Williams. In their series, roentgenograms of the sinuses were made on 61 of 64 patients with antrochoanal polyps and in only one instance were the results negative. In 35 cases (57.4%) there was evidence of a unilateral lesion on the involved side and 25 (42.6%) showed evidence of bilateral involvement of the maxillary or other sinuses. A tumor or mass could be demonstrated by roentgenogram in 7 (11.4%) of the 61 patients.

TABLE II

SHOWING SIDE OF INVOLVEMENT AND INTERPRETATION OF ROENTGENOGRAM

SIDE OF POLYP	INTERPRETATION OF ROENTGENOGRAM
Left	Opaque left antrum
Right	Opaque right antrum
Right	Opaque right antrum
Left	Bilateral maxillary sinusitis
Left	Thickening of mucosa, floor and lateral wall of left antrum
Right	Right antrum opaque. Appeared to contain large cyst.
Right	Right antrum opaque
Right	Right antrum opaque
Right	Choanal mass on right
Right	Cloudy ethmoid cells, bilateral
Left	Questionable cyst in left antrum
Left	Opaque left antrum; hazy left frontal and ethmoid. Rounded tumefaction in right antrum
Left	Opaque left antrum. Cloudy left ethmoid.

Interpretations of roentgenograms were available in only 13 of the series being reported. Table II shows the side of the nose involved and also the pertinent radiographic findings.

If a comparison is made with the statistics given by Heck, Hallberg, and Williams,[14] it will be seen that in this series a larger percentage (69.2%) showed involvement of one or more sinuses on the same side as the polyp and fewer (23.0%) showed bilateral involve-

ment. In one instance, a choanal mass was demonstrated by roentgenogram. In no instance did the roentgenograms fail to indicate a pathological condition.

Allergy. Although the numbers involved are small and therefore may not be of much significance, the records indicated that 5 (16.1%) of the patients gave a positive history of nasal allergy, hay fever, or asthma. Eleven records (35.4%) made no reference to an allergic background or noted any symptoms suggesting an allergy. In 15 instances (48.3%), a specific notation was made that there was no allergic background.

A differential blood cell count was noted in seven records (22.2%) and 11 (35.4%) had an eosinophilia of less than 3 per cent, while the remaining 6 (19.3%) had an eosinophilic cell count of 3 per cent or higher.

It is of interest to note that four of the patients with no history of an allergy had the highest eosinophilic count in the blood smear, viz., 3, 5, 10, and 17.5 per cent.

There were nasal smears reported on eight patients (25.9%); four of the smears showed no eosinophils and on each of the other four a few eosinophils were found.

Surgical Procedure and Pathology. In 24 cases in this series the polyps could be considered as typical in that they fulfilled the physical requirements of location, general appearance, etc., no evidence of a suppurative sinusitis was present, and the nasal cavity was free of additional polypoid growths. One of the 24 polyps was not a simple mucous growth but a polyp of the inverted papilloma type.

The histories indicated that 5 (20.8%) had had a choanal polyp removed previously with an average interval of two years between the first and second removal.

In this group of 24, the polyp was removed by snare and evulsion 19 times, the pedicle was cut twice, thereby freeing the polyp, and a Caldwell-Luc operation was performed 3 times.

There were seven patients, of the total of 31, in whom chronic suppurative sinusitis was proven. Four of these had recurrences; two after removal with the snare, one after a Caldwell-Luc procedure

(but with persistent suppurative sinusitis), and one after a polypectomy and intranasal antrotomy. The other 3 had radical surgery as the first procedure and there was no recurrence of the polyp.

The microscopic examination of the tissue removed from the sinuses by the external route showed, in every instance, chronic inflammation. Sections of the polyps removed by intranasal snare or avulsion were diagnosed as nasal polyp, and in many reports no mention was made of cellular infiltration.

It is admittedly difficult and perhaps improper to come to conclusions from a review of so few case histories but one cannot peruse the records without gaining certain impressions. From this study it would appear that the patient with a choanal polyp and chronic suppurative sinusitis is more likely to do better and is less likely to have a recurrence if a radical procedure, such as the Caldwell-Luc operation, is the initial one. If a choanal polyp exists in the absence of chronic suppurative sinusitis, and removal is accomplished by avulsion and snare, a recurrence can be anticipated in at least 20 per cent. This figure agrees fairly well with the incidence of recurrence following avulsion (26.6%) reported by Heck, Hallberg, and Williams. There was insufficient data to indicate the incidence of recurrence after the second removal.

Many of the reports on the origin of the choanal polyp which have been previously cited in this paper, leave the reader with the impression that once the interior of the antrum is inspected through a canine fossa opening, it is not difficult to find a stalk and to trace it into the middle meatus. This was not the case among the few of this series on whom radical sinus surgery was performed. In the group that had the sinus opened by the external route, in no instance was mention made of a pedicle in the description of the interior of the sinus. In one instance there was presumptive evidence that the growth arose from the medial wall of the antrum adjacent to a large accessory ostium.

In the descriptions of the interiors of the maxillary sinuses it was evident that marked hyperplasia, edema, cysts, and mucopus were observed, and it appeared that in this inflammatory mass of tissue it was practically impossible to discern a pedicle. If the patient had been seen early in the disease or before secondary involvement, it is quite possible that the stalk might be traced to its origin during a Caldwell-Luc procedure.

However, in this series the simple uncomplicated case was considered favorable for polypectomy by snare and avulsion and so the origin of the pedicle remained unknown.

SUMMARY AND CONCLUSIONS

It has been well established that the choanal polyp originates in a paranasal sinus, usually the maxillary, and extends on a pedicle from the middle meatus into the choana or nasopharynx, where it may continue to enlarge. Apparently it may arise from any wall of the maxillary sinus and gains entrance to the nasal fossa by way of an ostium, usually a large accessory ostium. It is predominantly solitary and unilateral. There are, as a rule, no other polypoid changes or polyps within the nose.

This particular polyp is considered by most observers to be the result of an inflammatory condition of the mucosa lining the sinus. In each instance that the maxillary sinus was opened in this series, the lining membrane was found to be thickened, polypoid, and edematous. The pathology report on the tissue removed in each instance was "nasal polyp and chronic inflammatory reaction."

Numerous authors have described a stalk within the maxillary sinus and stated that they have been able to locate the site of origin of the stalk and trace it to its exit from the antrum. In the sinuses that were opened in this series this was not accomplished, and the presence of edematous polypoid inflammatory tissue within the sinus would seem to make this a difficult observation.

The frequency of occurrence could not be determined with any degree of accuracy but the choanal polyp was encountered in almost every age group and is predominantly an adult disease.

The diagnosis should not be difficult if a careful examination is made of the nose and nasopharynx. Roentgen examination of the sinuses should always be included in the study of such cases since rarely are the results negative and helpful information may be expected.

The radical external sinus operation is probably the procedure of choice when there is chronic suppurative inflammatory disease of the lining membrane of the sinus from which the polyp arose. This review indicates, however, that good results may be expected in 80

per cent of persons without chronic sinusitis following removal of the polyp by snare and avulsion. Therefore, such a procedure may be justified in carefully selected cases.

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REFERENCES

1. Ash, J. E., and Raum, M.: An Atlas of Otolaryngic Pathology. Published jointly by: The American Academy of Ophthalmology and Otolaryngology, The American Registry of Pathology, and The Armed Forces Institute of Pathology; p. 159, 1955.

2. Bajkay, Tibor v.: Uber Choanalpolypen. Zeitschr. f. Laryn. Rhin., u. ihre Grenz. 24:321-326, 1933.

3. Baum, H. L.: Two Cases of Naso-Pharyngeal Polypus Originating in the Maxillary Sinus. Laryngoscope 28:180-186, 1918.

4. Citelli: Uber eine neue Krankneit der Oberkieferhohle. Archiv fur Laryngologie und Rhinologie 33:37-42, 1920.

5. Crowe, S. J.: The Nose; Chap. 3 in Lewis, D.: Practice of Surgery. Hagerstown: W. F. Prior Co. 4:1-76, 1948.

6. Crowe, S. J., and Baylor, J. W.: Benign and Malignant Growths of the Nasopharynx and Their Treatment with Radium. Arch. Surg. 6:429-488, 1923.

7. Davis, E. D. D.: Some Nasal Conditions. Brit. Med. Jour. 1:1167-1168, 1938.

8. Dawson, G. W.: A Case of Antro-Choanal Polypus of Exceptional Size. Lancet 1:1095-1096, 1922.

9. Dingley, A. R.: Some Observations on Antro-Choanal Polypus. Jour. Laryngol. and Otol. 63:141, 1949.

10. Ewing, Thomas: Antro-Nasal or Choanal Polypus. Jour. Laryngol. and Otol. 33:227-234, 1918.

11. Forschner, Leo: Zur Frage der Entstehung der Choanaepolypen. Monotschr. f. Ohren. u. Laryngo-Rhin. 55:1102-1108, 1921.

12. Guthrie, Douglas: Choanal Polypi in Children. Proc. Royal Soc. of Med. 12:153-155, 1918-19.

13. Hansel, French K.: Allergy of the Nose and Paranasal Sinuses. C. V. Mosby Co., St. Louis, p. 155, 1936.

14. Heck, W. E.: Hallberg, O. E., and Williams, H. L.: Antrochoanal Polyps. Arch. Otolaryn. 52:538-548, 1950.

15. Hirsch, Oskar: The Catarrhal Inflammation of the Nasal Accessory Sinuses and Its Diagnosis. Laryngoscope 37:1-9, 1927.

16. Hirsch, Oskar: Nasal Polypi: Their Origin and Its Solution. Bull. of the Battle Creek Sanit. and Hosp. Clinic 23-24:7-36, 1928-29.

17. Huizinga, E.: Een en ander over Choanaalpoliepen. Nederl. Tijdschr. o. Geneesk. 1:2433, 1927.

18. Kelly, A. Brown: A Contribution to the Pathology of the Antrum of Highmore. Glasgow Med. Jour. 62:279-286, 1904.

19. Kelly, A. Brown: Naso-Antral Polypus. Lancet 1:89-91, 1909.

20. Killian, Gustav: The Origin of Choanal Polypi. Lancet 2:81-82, 1906.

21. Kubo, Ino: Ueber die eigentliche Ursprungstelle und die Radikaloperation der Solitaren Choanal Polypen. Arch. F. Laryngol. u. Rhinol. 21:81-89, 1909.

22. Kubo, Ino: Weitere Studien uber die Solitaren Choanalpolypen aus den Nasennebenhohlen. 17th Internat. Congress of Medicine (London), 1913. Section 15, Part 2, pp. 423-426.

23. Laff, H. I.: Deforming and Recurring Polyps of Youth. Arch. Otol. 30:795-799, 1939.

24. Lang, K.: Einfache Radikaloperation der Solitaren Choanenpolypen 2:650, 1910.

25. Lang, K.: Fall eines Choanalen Polyps, Anthroskopie bei demselben. Zeitschr. f. Laryn. Rhinologie 5:240, 1913.

26. Lange, Victor: Zur operation der Choanalpolypen. Deut. Med. Wochenschr. 13:213-215, 1887.

27. Mangabeira-Albernaz: O Polipo De Killian na crianca. Revista oto-laringologica de Sao Paulo 6:361-371, 1938.

28. McKenzie, Dan: Discussion on Recurring Spheno-Choanal Polypus in a Child by Moore, I. Proc. Royal Soc. Med. 12:107, 1918-1919.

29. Meyers, David: Obstruction of the Nasopharynx Secondary to Choanal Polyp of Antral Origin. Arch. Otol. 44:328-333, 1946.

30. Meyerson, M. C.: The Ostium of the Maxillary Sinus. Trans. Amer. Laryng. Assn., 1931, pp. 146-159.

31. Michaelis, A.: Nasal Polypi in an Eight Year Old Child. Laryngoscope 38:776-777, 1928.

32. Mithoefer, W.: Hyperplastic Maxillary Sinusitis. Laryngoscope 36:137-146, 1926.

33. Moore, Irwin: Recurring Spheno-choanal Polypus in a Child. Proc. Royal Soc. of Med. 12:104-108, 1918-19.

34. Morwitz, S. M.: The Solitary Choanal Polypus. Ill. Med. Jour. 59:213-217, 1931.

35. Palfyn, Jean: Anatomie Chirurgicale, Paris, 1753.

36. Proetz, A. W.: Personal communication.

37. Ruysch, Fredrik: Observationum anatomico-chirurgicarum Centurca, 1691.

38. Sands, H. B.: Naso-Pharyngeal Polypus. N. Y. Med. Jour. 45:102, 1887.

39. Semeleder, F.: Rhinoscopy and Laryngoscopy. New York: Wm. Wood and Co., pp. 45-46, 1866.

40. Semenov, Herman: The Pathology of the Nose and Paranasal Sinuses in Relation to Allergy. Trans. Amer. Acad. Ophthal. and Otolaryn., pp. 121-170, 1952.

41. Shea, J. L.: A Report of Two Intranasal Angiomas, and an Unusually Large Antral Polyp. Trans. Am. Laryng. Rhin. and Otol. Soc. 36:465-472, 1930.

42. Stawraski, S.: Zur Frage uber die Herkunft der retronasalen (Choanal) polypen. Monatschr. f. Ohrenh. u. Laryngo-Rhinol. 62:1147-1152, 1928.

43. Syme, W. S.: Choanal Polypus. J. Laryng. and Otol. 31:515-518, 1916.

44. Syme, W. S.: A Series of Cases of Maxillary Antral Disease. Proc. Royal Soc. of Med. 12:255, 1918-19.

45. Thomson, St. Clair, and Negus, V. E.: Diseases of the Nose and Throat. New York: Appleton, Century, Crofts Co., 5th Ed., pp. 175-177, 1947.

46. Voorhees, I. W.: Large Postnasal Polyp in Child. Arch. Pediat. 50:367-368, 1933.

47. Waldapfel, Richard: Choanal Polypi. Eye, Ear, Nose, and Throat Monthly 30:79-80, 1951.

48. Walsh, T. E., and Lindsay, J. R.: Cytology of Nasal Polypi. Arch. Otolaryngol. 20:649-658, 1934.

49. Wishart, D. J. G.: The Origin of the Naso-Antral Polyp. Canada M. A. J. 6:7-11, 1916.

50. Zuckerkandl, E.: Normale u. Pathologische Anatomie der Nasenhohle. Vienna: Braumuller, 1892.

XXIII

HEAT AND MOISTURE EXCHANGE OF RESPIRATORY MUCOUS MEMBRANE

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From the earliest studies of nasal physiology, it was concluded that the prime function of the nasal mucous membrane was to prepare inspired air for efficient oxygen exchange in the lung alveoli. As Proetz[1] succinctly put it, the nose is the air conditioning unit of the respiratory tract. Perwitzschky[2] carried out an exhaustive study of temperature and humidity changes in the air passages. Inspired air was shown to reach a temperature of 32° C. (89.6° F.) and a relative humidity of 79 per cent in the nasopharynx. Relative humidity in the trachea of a cannulized patient reached only 51 per cent. Only after the relative humidity of the inspired air was raised to 80 per cent did the air in the trachea agree with the air in the nasopharynx.

It was not until recent comparative anatomy studies of the nasal interior were made that new concepts in the nasal functions were evolved. Scott[3] has shown that certain arctic aquatic mammals like the seal have extremely complex turbinate structures for body heat regulation. Jacquot and Mayer[4] showed that with the rabbit, which has a complex maxillary turbinate, the water content of expiratory air varied between 17 and 38 mgm per liter when the animal continued to breathe room air and its body exposed to cold and hot environments, respectively. There can be little doubt that the nasal mucous membrane is well suited for humidification and heating of inspired air but this, phylogenetically speaking, could well be a secondary function. Cole[5] also proposed that the intranasal structures were primarily concerned with regulating body heat.

The purpose of this presentation is to attempt to shed more light on this concept. The air conditioning capacity of the nasal mucous membrane will be compared with that of the trachea. Further comparisons will be made with a simple model simulating an adult trachea.

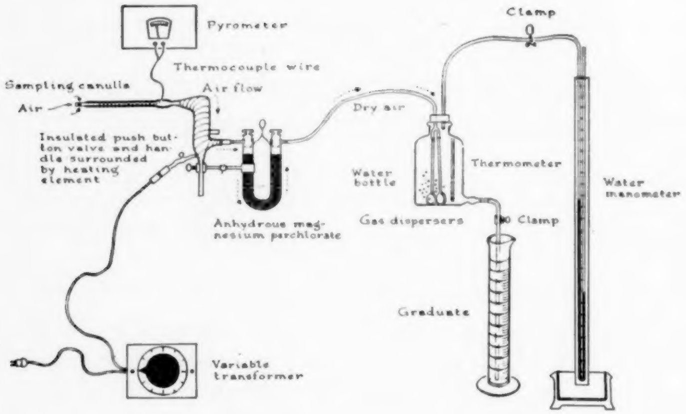

Fig. 1.—Schematic diagram of apparatus assembled and ready for sampling.

APPARATUS

The apparatus employed in these experiments is essentially that developed by Lauren Seeley[6] in 1940. A schematic diagram of the assembled apparatus is shown in Figure 1 and a photograph is shown in Figure 2.

The sampling cannula (A) is a five-inch long malleable chrome plated copper tube with an external diameter of 4 mm and a lumen diameter of 3 mm. The open ended cannula has two half-millimeter sized holes drilled through the sides just above the distal end. Fine iron constantin thermocouple wire (K) was threaded through an opening in the side and fixed in position by sealing the opening with waterproof cement. The thermocouple junction within the tube is located one inch above the tip. The entire tube was sheathed in rubber tubing giving a total external diameter of four and one-half mm.

(B) is a commercial push button valve modified to meet the requirements of the experiment. Surrounding the copper valve is a heating element constructed from high resistance wire insulated with glass tubing and held in place with adhesive tape. A variable resistance transformer (G) controls the current flowing through the heating element.

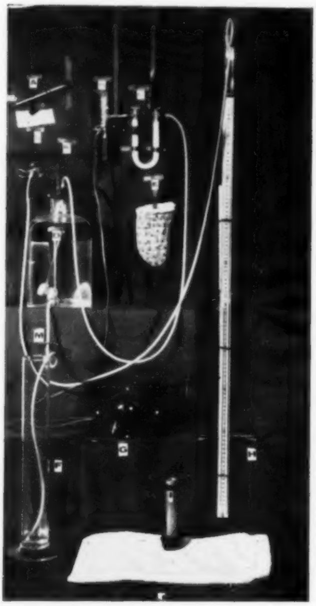

Fig. 2.—Photograph of assembled apparatus.

(C) is a Schwartz 150 mm drying tube containing about 100 grams of anhydrous magnesium perchlorate. The drying tube is handled through an insulated bag (E). A rectangular piece of lamb's wool (L) is used to further insulate the portion of the sample tube that lies outside the nose.

(D) is a 4000 cc glass collecting bottle filled with distilled water. Two gas dispersion tubes of coarse porosity reach the bottom of the bottle. These are connected to the drying tube by rubber tubing. A laboratory mercury thermometer (not shown) is kept submerged in the bottle. A water manometer (H) is attached to the collecting bottle by rubber tubing interrupted by a clamp. The aspirator type collecting bottle is constructed with a glass tube near the bottom. The 1000 cc graduate (F) is set about three feet below the collecting bottle and receives the rubber tubing through which the water flows from the collecting bottle.

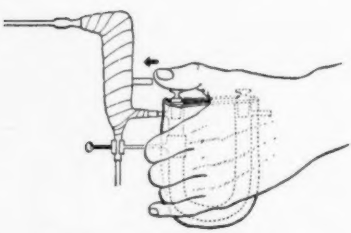

Fig. 3.—Diagram showing attachment of drying tube to sampling push button valve and sampling cannula.

(J) is a commercial heating pad. (I) is a piece of glass tubing measuring five and one-half inches in length and one inch in diameter (average size of an adult trachea). The tube is lined with 1 mm thick blotting paper. The sampling tube, push button valve, and drying tube are handled as a unit although the three elements can be detached from each other. The push button valve and drying tube are held together rigidly by a burette clamp.

METHOD OF OBTAINING SAMPLES

In general, the sample of air is collected as follows: the drying tube is weighed the same day the experiment is performed, preferably just before the sample is taken, and placed in an insulated bag. One arm of the U tube is attached to the push button valve fitting by a very short piece of plastic tubing. It is very important that the tubing serve only to hold the connection together. Every effort is made to keep the fittings in close contact. This precaution is taken in order to avoid condensation of water in the plastic tubing. A small segment of tubing measuring three-fourths of an inch was found to condense 3 milligrams of moisture when the room temperature was 51° F. When one considers that we are dealing with amounts of water as small as 18 milligrams per 1000 cc of air, it is understandable why attention to this detail is important. The burette clamp which is kept permanently attached to the push button valve is now fastened to one arm of the U tube. This facilitates handling of the apparatus as shown in Figure 3. The heating element is turned on and regulated to the desired temperature (100° F.). After attaching the sampling cannula securely to the push button valve, the entire unit of cannula, valve, and drying tube are kept warm on a heating pad. Just before

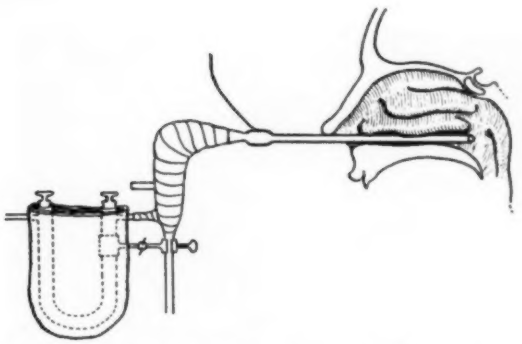

Fig. 4.—Diagram showing position of sampling cannula in nose.

the sampling is begun the rubber tubing from the collecting bottle is attached to the glass fitting on the drying tube and the stop cocks on the latter are opened. If no air bubbles through the gas dispersers in the collecting bottle, we are assured that the system is leak proof, at least to the level of the push button valve. The sampling cannula is then inserted into the subject. The desired distance has been previously determined and marked on the cannula with a thin strip of adhesive tape. The air sample is collected by opening the push button valve during the desired respiratory phase. The movement of air through the system is made possible by the difference in level between the water in the collecting bottle and the water in the graduate. The volume of water collected in the graduate is equivalent to the amount of air removed from the respiratory tract. During each sampling, 800-1000 cc of air is collected. The purpose of the gas dispersion tube was to assure complete saturation of the dry air by the water in the collecting bottle. Immediately after the sample is collected, a manometer and temperature readings are taken, the volume of water in the graduate is measured, and the stop cocks on the dessicating tubes are closed. In the meantime, the room air temperature, relative humidity, and air sample temperatures are being recorded automatically. The drying tubes are taken to the balance room, allowed to adjust to room temperature, and weighed. A barometric pressure reading is recorded just prior to the sampling.

ENVIRONMENTAL ROOMS AND ENVIRONMENTAL CONDITIONS

The subjects were observed and the air samples collected in two air conditioned rooms in which temperature and humidity could be accurately controlled. These rooms measured 12 feet long, 9 feet

Fig. 5.—Photograph showing an air sample being taken from nose.

wide, and 8 feet high, with the heating and cooling equipment located on the floor above. Through perforations in the ceiling the conditioned air is brought into the room. An observation chamber is located between the testing rooms where self-recording aspirating psychrometers are placed. Sling psychrometric readings were taken during the tests to check the self-recording psychrometers.

PROCEDURE

The subject entered the test room at least 30 minutes before samples were taken. He was seated about three feet from the psychrometer. Nasal samples were obtained from one subject whose nasal anatomy and physiology were considered normal. The second subject had a laryngectomy and right radical neck dissection done three months previously. He had an excellent stoma measuring about one inch in diameter. No tracheotomy tube had been worn for at least two months. The tracheal lumen could be visualized as far as the bifurcation. Before insertion of the sampling cannula, the floor of the nose was anesthetized lightly by passing a cotton tipped applicator impregnated with one-half per cent pontocaine hydrochloride to the posterior nares. The trachea was lightly anesthetized by spraying the same solution once or twice through the tracheostomy.

Inspiratory and expiratory samples were taken during the latter part of each respiratory phase in order to allow for clearing of the

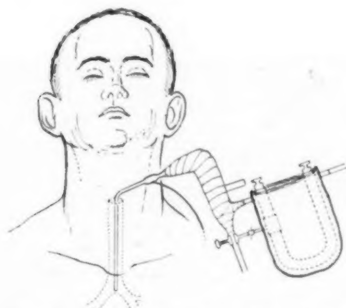

Fig. 6.—Diagram showing position of sampling cannula at carina.

air from the preceding phase. The respiratory rate was about 12 per minute with almost a complete complement of air taken at each breath. About 15 cc of air could be collected during each cycle.

The inspiratory and expiratory nasal air samples were taken from just beyond the posterior choana (Figs. 4, 5). Additional expiratory samples were taken at the external nares. The tracheal samples were drawn from just above the carina (Figs. 6, 7). The cannula was held in the center of the lumen under direct vision. The tip of the cannula must be watched constantly to avoid contamination with an occasional accumulation of mucus. Additional expiratory samples were taken at the tracheal stoma.

A third set of experiments was carried out on a glass tracheal model having the dimensions of an "average" trachea (Fig. 8). This tube is lined with blotting paper and the latter is saturated with distilled water. The tube is warmed by an encircling heating element. One end of the model is placed at the mouth and the sampling cannula is inserted through the opposite end and reaches within one-half inch of the lips. Ambient air is drawn into the tracheal model by inspiring through the mouth at the rate of 12 times per minute. Expiration is carried on through the nose.

Each subject was studied at three different environmental conditions. The dry bulb temperatures were 51° F., 72° F., and 80° F. with relative humidities of 65 per cent, 45 per cent, and 25 per cent, respectively. A total of 69 individual samplings was analyzed.

An increase in weight of the drying tube after sampling represents the amount of moisture in the given volume of air collected.

Fig. 7.—Photograph showing an air sample being taken from trachea.

The number of grains of moisture per pound of dry air (absolute humidity) was calculated and reduced to standard conditions. Changes in temperature and absolute humidity were calculated and finally, relative humidities determined.

During the course of the experiments the absorption method was checked against the psychrometer by taking samples of room air at 90-95° F. and 85 per cent relative humidity. Although the absorption method gave slightly lower readings, the differences were considered insignificant. Seeley reported a similar experience with the two methods.[6]

RESULTS

Table I shows the temperature and moisture content of air inspired at a temperature of 72° F. and 45 per cent relative humidity when it reaches the posterior nares in a normal nose. The temperature of 90° F. and absolute humidity of 175 grains of water vapor per pound of dry air (80 per cent relative humidity) is compared with a temperature of 85° F. and 173 grains of water per pound of dry air (90 per cent relative humidity) when air reaches the tracheal bifurcation in a laryngectomized and decannulated patient. These can be further compared with a glass tube equal in length and diameter to the adult trachea, lined with water saturated blotting paper and warmed to 85° F. The latter gives up 175 grains of

TABLE I

INSPIRATION

Room Dry Bulb Temperature 72° F.
Room Relative Humidity 45%
Room Absolute Humidity 52 grains/lb. dry air

	TEMPERATURE	NET TEMP. CHANGE	ABSOLUTE HUMIDITY GRAINS/LB. DRY AIR	NET WATER CHANGE GRAINS/LB. DRY AIR	RELATIVE HUMIDITY
Nasal Air at Choana	90° F.	+18° F.	175	+123	80%
Tracheal Air at Bifurcation (laryngectomized)	85° F.	+13° F.	173	+121	90%
Tracheal Model Air at Bifurcation	85° F.	+13° F.	175	+123	95%

water vapor per pound of dry air and humidifies the air to within 5 per cent of saturation (95 per cent relative humidity).

Table II shows that the expired air reaches an average temperature of 94° F. and attains an absolute humidity of 213 grains of water vapor per pound of dry air as it passes the posterior nares. As the air flows forward toward the anterior nares the mucous membrane

TABLE II

EXPIRATION

Room Dry Bulb Temperature 72° F.
Room Relative Humidity 45%
Room Absolute Humidity 52 grains/lb. dry air

	TEMPERATURE	NET TEMP. CHANGE	ABSOLUTE HUMIDITY GRAINS/LB. DRY AIR	NET WATER CHANGE GRAINS/LB. DRY AIR	RELATIVE HUMIDITY
Nasal Air at Choana	94° F.	+22° F.	213	+161	85%
Nasal Air at Nostril	86° F.	+14° F.	193	+141	95%
Tracheal Air at Bifurcation	91° F.	+19° F.	212	+160	92%
Tracheal Air at Stoma	86° F.	+14° F.	187	+135	96%

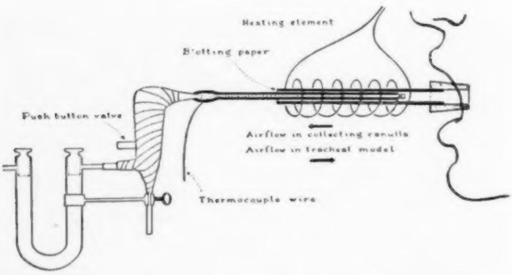

Fig. 8.—Diagram showing glass tracheal model with sampling cannula in position.

recovers 20 grains of moisture and an equivalent amount of heat, the temperature dropping to 86° F. The temperature of the expired air at the bifurcation of the isolated trachea is 91° F. and contains 39 grains of moisture per pound of dry air in excess of that present in the inspired air (Table I). Twenty-five grains are recovered by the time the air reaches the stoma with the temperature dropping to 86° F.

Tables III and IV show the results in an ambient atmosphere of 51° F. and 65 per cent relative humidity.

Tables V and VI show the results obtained when the inspired air was 80° F. at 25 per cent saturation.

Figures 9 to 14 represent the above results in graphic form.

COMMENT

Mechanism of Heat and Moisture Exchange. As air passes over a moist surface like the respiratory mucous membrane, an exchange of heat and moisture takes place with amazing rapidity. If the air is drier and cooler than the wet surface, as is usually the case during inspiration, water and heat will be extracted from the surface and added to the inspired air. Moisture is supplied by the secretion of mucus from the tuboalveolar glands in the respiratory mucous membrane. Heat is transmitted by the rich network of blood vessels in the submucosa. Both structures are regulated by nerve fibers from the autonomic nervous system, which carry impulses initiated in various parts of the body.

The transference of water from the mucous film on the surface of the respiratory mucosa to the air passing over it depends upon

TABLE III

INSPIRATION

Room Dry Bulb Temperature 51° F.
Room Relative Humidity 65%
Room Absolute Humidity 36 grains/lb. dry air

	TEMPERATURE	NET TEMP. CHANGE	ABSOLUTE HUMIDITY GRAINS/LB. DRY AIR	NET WATER CHANGE GRAINS/LB. DRY AIR	RELATIVE HUMIDITY
Nasal Air at Choana	90° F.	+39.0° F.	185	+149	85%
Tracheal Air at Bifurcation (laryngectomized)	84° F.	+33.0° F.	173	+137	95%
Tracheal Model Air at Bifurcation	85° F.	+34.0° F.	173	+137	96%

TABLE IV

EXPIRATION

Room Dry Bulb Temperature 51° F.
Room Relative Humidity 65%
Room Absolute Humidity 36 grains/lb. dry air

	TEMPERATURE	NET TEMP. CHANGE	ABSOLUTE HUMIDITY GRAINS/LB. DRY AIR	NET WATER CHANGE GRAINS/LB. DRY AIR	RELATIVE HUMIDITY
Nasal Air at Choana	94° F.	+43.0° F.	214	+178	83%
Nasal Air at Nostril	85° F.	+34.0° F.	187	+151	96%
Tracheal Air at Bifurcation	91° F.	+39.0° F.	205	+169	90%
Tracheal Air at Stoma	88° F.	+37.0° F.	200	+164	97%

certain interrelated physical factors such as temperature, moisture, speed of movement of air, presence of various substances which affect the osmotic pressure, etc. The process which results in surface water passing from the liquid into the vapor state is known as evaporation. A certain amount of heat is necessary to affect this transfer (heat of vaporization).

The tendency of water to pass into vapor depends upon the pressure of the molecules of water vapor over the surface of the water

TABLE V

INSPIRATION

Room Dry Bulb Temperature 80° F.
Room Relative Humidity 25%
Room Absolute Humidity 47 grains/lb. dry air

	TEMPERATURE	NET TEMP. CHANGE	ABSOLUTE HUMIDITY GRAINS/LB. DRY AIR	NET WATER CHANGE GRAINS/LB. DRY AIR	RELATIVE HUMIDITY
Nasal Air at Choana	90.5° F.	+10.5° F.	185	+138	83%
Tracheal Air at Bifurcation (laryngectomized)	86.5° F.	+ 6.5° F.	174	+127	89%
Tracheal Model Air at Bifurcation	85° F.	+ 5° F.	181	+134	98%

TABLE VI

EXPIRATION

Room Dry Bulb Temperature 80° F.
Room Relative Humidity 25%
Room Absolute Humidity 47 grains/lb. dry air

	TEMPERATURE	NET TEMP. CHANGE	ABSOLUTE HUMIDITY GRAINS/LB. DRY AIR	NET WATER CHANGE GRAINS/LB. DRY AIR	RELATIVE HUMIDITY
Nasal Air at Choana	95° F.	+15.0° F.	215	+168	85%
Nasal Air at Nostril	86.5° F.	+ 6.5° F.	193	+146	96%
Tracheal Air at Bifurcation	91° F.	+11.0° F.	214	+167	93%
Tracheal Air at Stoma	88° F.	+ 8.0° F.	200	+153	97%

(vapor pressure). Vapor pressure increases in proportion to the temperature and varies with the nature of the liquid. For example, certain dissolved or suspended substances will decrease the vapor pressure of water. Mucus, which is water altered in such a manner, cannot moisten air as easily as pure water.

The amount of moisture present in a given volume of air is known as absolute humidity. It is usually expressed in pounds or

grains per pound of dry air. The amount of moisture that a given volume of air can hold is not always the same, but will vary with the temperature. The amount of moisture which the air can hold varies directly with the temperature, but not proportionately. To show this relationship of temperature to moisture holding power, the term relative humidity is used. Relative humidity is the amount of moisture that air contains at a certain temperature, compared to what it could hold were it completely saturated at that temperature. Relative humidity is usually expressed in percentage:

$$\text{R.H.\%} = \frac{\text{Amount of water vapor present}}{\text{Amount of water at saturation}} \times 100$$

For example in Table I, we find that on inspiration there are 175 grains of moisture per pound of dry air at 90° F. in the nasopharynx. This gives a relative humidity of 80 per cent. At the bifurcation of the trachea, where the moisture content is about the same, namely, 173 grains of moisture per pound of dry air, at 85° F., the relative humidity is 90 per cent.

The heat lost by the respiratory mucous membrane is brought about by three processes of heat transfer; namely, convection, radiation, and evaporation. Mucous membrane differs from skin in the matter of heat loss in the fact that the former is always moist while the latter is moist only at times. Nevertheless, the similarity is quite apparent suggesting the possibility of a common function.

Interpretation of Findings. Table I shows that air which contains 52 grains of moisture at 72° F. gets an additional 123 grains from the trachea when it passes directly over the mucous membrane. A tube lined with moist blotting paper and heated so that the air in the tube lumen is 85° F. can yield the same amount of moisture. These figures compare favorably with air which has passed through the nose to the posterior nares. The trachea was able to warm the air to 85° F. while the nose which is considered a highly efficient air conditioning device could do little better. When the inspired air was 51° F. the trachea could add sufficient heat to raise the temperature to 84° F., an increment of 33°. The nasal mucosa could increase the temperature only 5° more. The moisture increment in the nose was only 12 grains per pound of dry air greater than in either the trachea or tracheal model. In a relatively dry atmosphere of 80° F. and 25 per cent relative humidity, the trachea yielded 127 grains of moisture compared with 138 grains for the nasal chambers. The blotting paper with 134 grains performed about as well as the nasal mucosa.

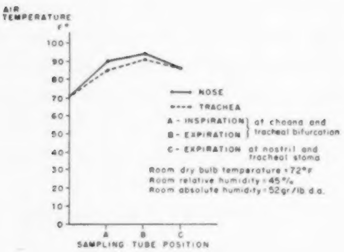

Fig. 9.—Comparison of changes in temperature as air passes through nose versus trachea (tracheostomy).

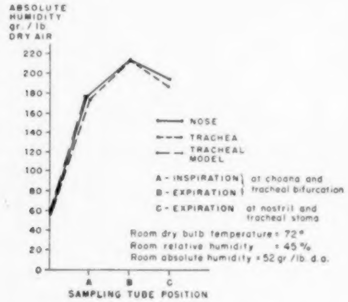

Fig. 10.—Comparison of water vapor changes as air passes through nose versus segment (tracheostomy) and tracheal model.

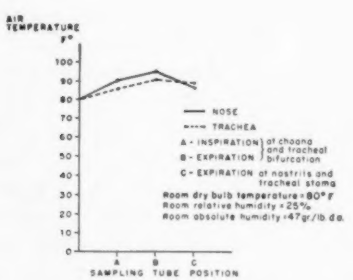

Fig. 11.—Comparison of changes in temperature as air passes through nose versus trachea (tracheostomy).

The above indicates that much simpler structures can humidify and warm air about as well as the human mucous membrane. This suggests the possibility that the moisture and heat exchange may serve another purpose besides conditioning air for pulmonary gas exchange.

Scott[3] comparing the anatomy of the nasal chambers in various mammals with that in the human, has shown that man has a relatively simple turbinate structure with a reduced area of mucous membrane. This he attributes to the fact that man has developed the skin into the chief organ of heat loss by shedding his hair and developing the sweat glands and subepithelial capillary plexuses. The more active hairy creatures in the animal kingdom have the most highly developed maxillary turbinates because they depend upon this organ for the loss of accumulated heat (seal, dog). The cat which is largely a sedentary animal whose activity is limited to short periods has a rather simple maxillary turbinate. The whale, whose skin is so constructed that the heat loss is efficiently prevented, has no turbinate structures, yet the inspired air is adequately conditioned.

Speculations. Despite this apparent evolutionary regression of human intranasal development, the nose has been left more than adequately endowed to meet the requirements of respiratory air conditioning from the coldest to the hottest climates. However, as man migrated to less temperate parts of the globe, he found it necessary to protect himself against inclemency. The result has been the modern heated home and apartment house with an atmosphere approaching that of the Sahara Desert in moisture content. During the winter months the amount of moisture per unit volume of outdoor air is very small despite its high relative humidity. In most northern cities the relative humidity of outdoor air is depressed to less than 25 per cent when it is warmed to 72° F. Seeley[6] has shown experimentally that going from air at 18.9° F. with a relative humidity of 57.5 per cent into a room of 70° F. at 20 per cent relative humidity the evaporation from the nasal mucosa changes from 112 grains to 168 grains of vapor per pound of dry air, an increase of 46 per cent. This could happen on a cold winter day. The change in rate of evaporation takes place with almost instantaneous rapidity, resulting in an alteration in the physical characteristics of the nasal mucous film. The viscosity, surface tension, and osmotic pressure of the mucus is increased. The consequences of this drying effect upon ciliary activity has been repeatedly demonstrated by Proetz.

Thus we find ourselves in somewhat of a dilemma because of man's constant attempt to thwart nature. By his ingenuity he has

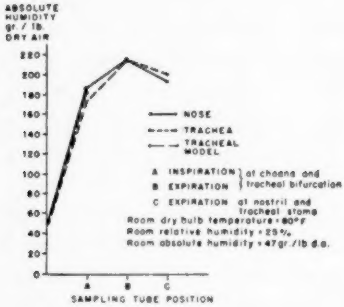

Fig. 12.—Comparison of water vapor content changes as air passes through nose versus tracheal segment (tracheostomy) and tracheal model.

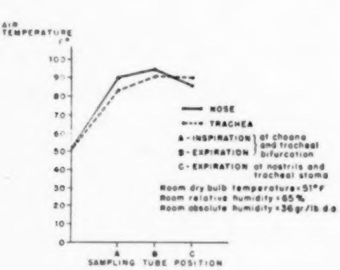

Fig. 13.—Comparison of changes in temperature as air passes through nose versus trachea (tracheostomy).

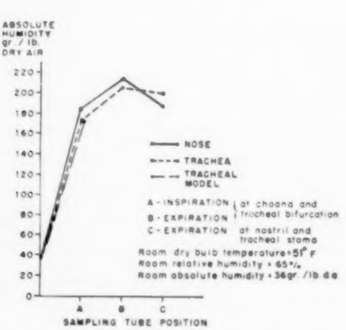

Fig. 14.—Comparison of water vapor changes as air passes through nose versus tracheal segment (tracheostomy) and tracheal model.

made it possible to live in every corner of the earth but finds himself frustrated by what seems to be a simple problem. As Proetz[7] aptly put it: "Medical programs are packed with brilliant last words on every subject from broad spectrum antibiotics to sines and cosines of the nasal angle while here I stand still wondering how to moisten a house."

SUMMARY

1. The concept has been presented that the nasal mucous membrane was primarily concerned with body heat regulation and only secondarily as an air conditioner.

2. Experiments were carried out which indicated that under ordinary conditions a laryngectomized patient could handle inspired air as efficiently as a person having a normal nasal anatomy and physiology. A warm moist tube of blotting paper could humidify ambient air to the same degree as the trachea or nose.

3. The physical changes that occur when air passes over moistened respiratory surfaces were discussed.

4. By reviewing the comparative anatomy studies of James H. Scott further evidence was presented that the primary function of the nose was heat regulation.

5. Attention was again brought to the dilemma of modern heating systems which create an atmosphere detrimental to the well-being of our respiratory tract.

1021 PROSPECT AVE.

REFERENCES

1. Proetz, A. W.: Semon Lecture: Respiratory Air Currents and Their Clinical Aspects. J. Laryng. and Otol. 67:1, 1953.

2. Perwitzschky, R.: Temperature and Moisture of Air and Air Passages. Arch. Ohr. Nas., a. Kehlkopf 117:1, 1927.

3. Scott, J. H.: Heat Regulating Function of the Nasal Mucous Membrane. J. Laryng. and Otol. 68:308, 1954.

4. Jacquot, Rand and Mayer, A.: Polypnée thermique et teneur en eau de l'air expiré. Soci. Biol. 23:1471, 1925.

5. Cole, P.: Some Aspects of Temperature, Moisture and Heat Relationship in Upper Respiratory Tract. J. Laryng. and Otol. 67:449, 1953.

6. Seeley, Lauren E.: Study of Changes in Temperature and Water Vapor Content of Respired Air in Nasal Cavity. Heating, Piping and Air-conditioning 51:337, 1940.

7. Proetz, A. W.: Humidity, a Problem in Air Conditioning. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 65:376, 1956.

XXIV

OBSERVATIONS ON THE EXCHANGE OF FLUID IN THE NOSE AND RESPIRATORY TRACT

Sir Victor Negus

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The problem is to determine how the various sources of fluid derived from the atmosphere, or from tissues of the body by transudation or secretion, are regulated so accurately as to maintain a layer of moisture on the mucosa of the nose and respiratory tract, but without flooding the lumen. The necessity for this covering of moisture is a legacy of an aquatic origin.

The objects of humidification are the entrapping of olfactory molecules, provision of a liquid covering for ciliary action, maintenance of respiratory exchanges in the terminal air sacs and lubrication of the vocal folds.

First it is desirable to study two physical processes, transudation of fluid by hydrostatic pressure and active transference by osmosis.

The mucous membranes concerned vary from the squamous epithelium lining the anterior third of the nose of most dogs, the columnar ciliated covering of the septum, maxilloturbinal and bronchial passages of most mammals and the thick olfactory epithelium devoid of cilia, to the extremely thin membrane, two cells thick, covering the maxilloturbinals of rabbits and the still thinner lining of the air sacs and alveoli, a single cell in thickness.

Under some of these membranes there is a system of vascular spaces, under sympathetic control to supply the requisite amount of warmth and transudate (Fig. 1).

HYDROSTATIC PRESSURE

This is the ordinary process of ultra filtration[4] by which plasma in the capillaries exudes through the freely permeable walls, but with-

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out the larger protein molecules, to fill the cell and tissue spaces with serum; the pressure in the arterial end of the capillary loop is about 40 cm H_2O and at the venous end about 17 or only 4 or 5 cm H_2O, while in the tissues it is about 20 mm H_2O (Fig. 2).

A great deal of fluid is reabsorbed at the venous end by osmotic pressure.[16] The capillary walls at the venous end are much the more permeable,[4] with larger pores.[20]

Not all the cells of the epithelium and connective tissue are in contact with capillaries and consequently tissue fluid is essential for the processes of metabolism; the cells play an active part in the transference of fluid, particularly in the intestines.

The lymphatics drain away much fluid, their walls being more permeable than the corresponding blood capillaries, with a less resistant cement material.

DIFFUSION

The movement of solutes or molecules in solution causes the solutes to travel within the liquid from places where they are more concentrated to parts where they are more dilute, until a uniform concentration is finally attained.

The process appears to have some application in the present inquiry in relation to passage of fluid out of the capillaries, which is said to occur much more rapidly by diffusion processes than by hydrostatic pressure.[20]

CELL MEMBRANES

The membrane of a cell is constituted by an aggregation of protoplasm with the assistance of certain salts and proteins adsorbed on the external surface; the surface film is restored if punctured, being a condensation layer of which calcium is an essential constituent.[16]

These walls are so thin that nothing would appear to prevent excessive transudation except the equilibrium that is contantly maintained in fluid exchanges by outpouring from capillaries, with constant reabsorption; since there is an active pressure in the tissue fluids, however low, there is always the probability of escape of moisture from the free epithelial surface (Fig. 2).

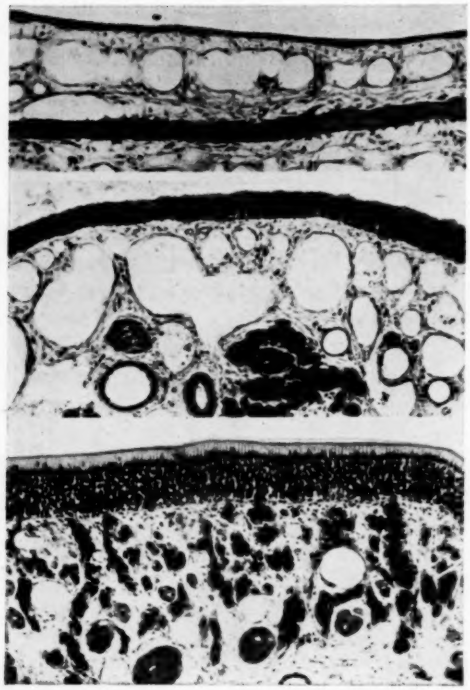


Fig. 1.—Rabbit's nasal mucosa, ⅔ Objective, to show the subepithelial vascular spaces. Top: stratified; middle: ciliated; bottom: olfactory.

In the membranes are pores of varying size; it is mainly through the intercellular regions that the bulk of fluid exchange, including water and dissolved electrolytes, appears to pass.[20]

FLUID AND GASEOUS EXCHANGES IN THE ALVEOLI OF THE LUNG

Histological structure is simple, there being a very thin epithelial lining,[4] demonstrable by electron microscopy,[2] with a single layer of big flat cells in contact with the single celled endothelial walls of the capillaries; there are no cilia and no goblet cells or multicellular glands (Fig. 3).

Oxygen and CO_2 pass through the epithelial layer directly into or out of the capillaries, provided the lining of the alveoli remains

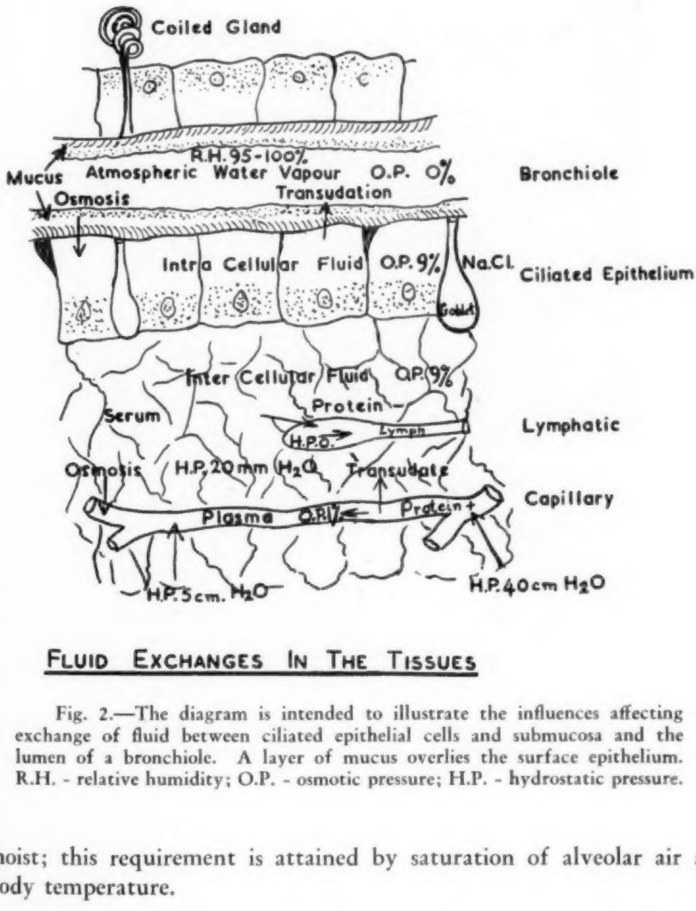


Fig. 2.—The diagram is intended to illustrate the influences affecting exchange of fluid between ciliated epithelial cells and submucosa and the lumen of a bronchiole. A layer of mucus overlies the surface epithelium. R.H. - relative humidity; O.P. - osmotic pressure; H.P. - hydrostatic pressure.

moist; this requirement is attained by saturation of alveolar air at body temperature.

It is unlikely that any fluid exchange takes place between the alveoli and the adjoining capillaries, since complete humidification is provided for in the respiratory tract before the atria are reached; it is the nose and upper respiratory tract, and not the alveoli, which supply moisture.

Hydrostatic pressure in the pulmonary capillaries is extremely low but appears to be sufficient to balance the potential osmotic pressure between that exerted by alveolar air, of no ionic content, and plasma with an osmotic pressure equal to 0.9 per cent NaCl; since

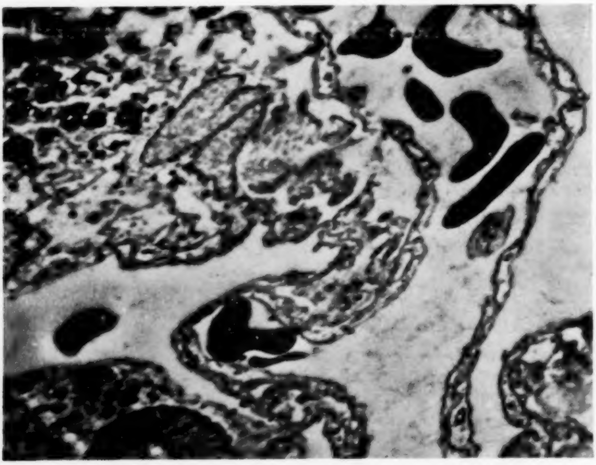


Fig. 3.—Photomicrograph taken with the electron microscope to show the relation of capillaries and alveoli. Two capillaries are seen, that on the left containing a section of a red blood corpuscle and that on the right having sections of six corpuscles in its lumen. On the extreme right an alveolus is shown; the wall separating it from the capillary has an epithelial and an endothelial layer. (From the department of Anatomy, Royal College of Surgeons of England, by kind permission of Professor Causey. X 4,700 in the original).

alveolar air is at body temperature there is, of course, no possibility of condensation (Fig. 7).

OSMOTIC PRESSURE AND ITS REGULATION

A semipermeable membrane is one which allows passage of water but not of solute from the side of weaker concentration to the stronger of two solutions.[1]

There are no perfect semipermeable membranes in the animal body, but there are many which exert a selective action, as in the retention of large protein molecules in capillaries.

Some simple organisms such as *Noctiluca*, a flagellate, are in osmotic balance with the medium in which they live; this particular organism lives in sea water and if transferred to a more dilute medium it swells up and bursts.[14]

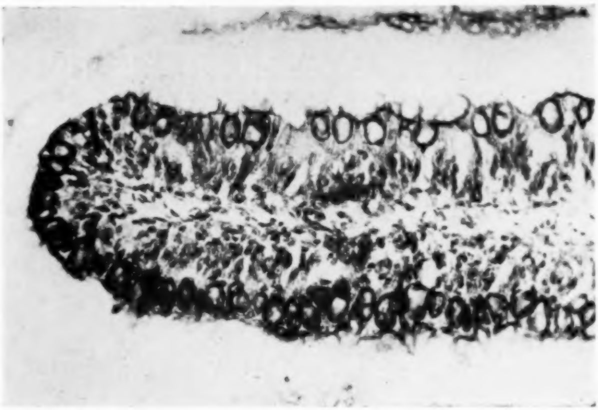


Fig. 4.—High power view of a lamella from the olfactory recess of a Carp. The goblet cells are large and numerous and some are being discharged.

Fish and some aquatic amphibia have the problem of osmotic pressure to contend with. Their blood plasma and tissue fluids correspond to a concentration of 0.9 per cent NaCl; sea water is roughly equivalent to 3.4 per cent NaCl, while river water has a very low ionic content (Fig. 5).

These aquatic species, being subject to the force of osmotic pressure, are in constant danger of flooding of the tissues when in fresh water and of dessication while in the sea.[1]

Permeability of the skin is reduced, partly by its density, but also, in many species, by reinforcement with a layer of mucus, the mucin of which is insoluble in water; excess of fluid intake is counteracted by increased output by the kidneys.[14]

The olfactory recess of fish, being lined by delicate epithelium, is an especially permeable region; on the projecting lamellae there are numerous goblet cells which discharge mucus on the surface to provide a covering, presumably as a waterproofing layer (Fig. 4).

IONIC COMPOSITION OF BODY FLUIDS

Primitive blood probably consisted of little but plain sea water capable of carrying sufficient dissolved oxygen.[1] The blood of verte-

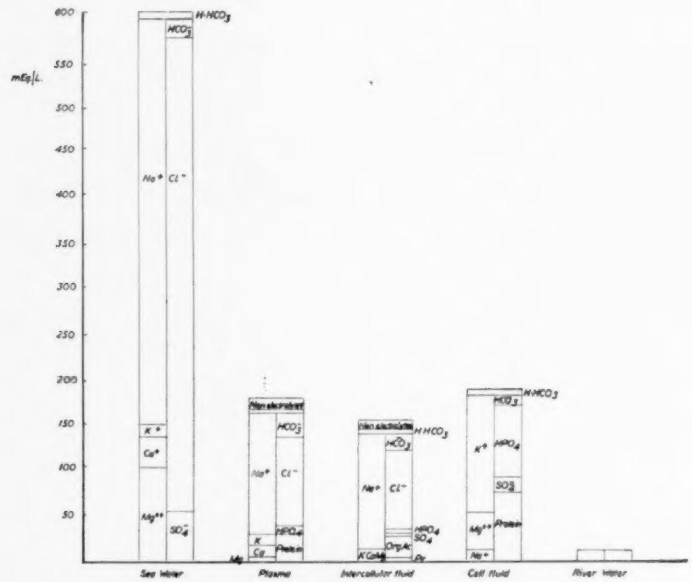


Fig. 5.—Diagram to illustrate the relative ionic content of sea water, river water, blood and tissue fluids. (Adapted from Gamble, J. L., 1951. Lane Medical Lectures, Vol. 5, No. 1, Stanford University Press.)

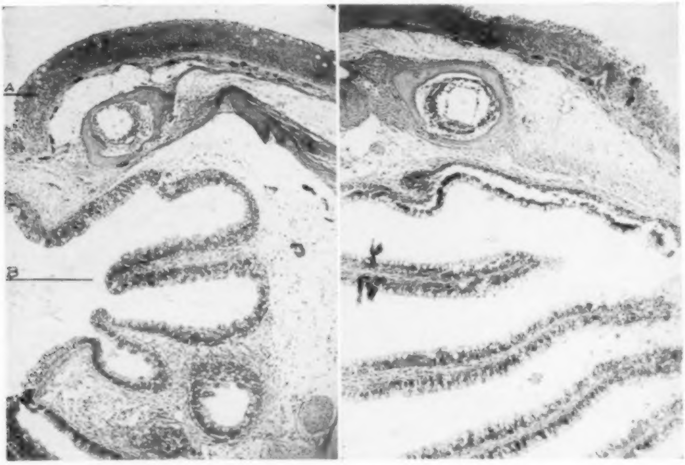
brates resembles sea water in ionic composition but its concentration is approximately three times less. The plasma of the blood, the tissue fluid within body cells and that in the intercellular spaces are all in ionic balance; blood contains more protein[10] (Fig. 5).

In man slightly over two-thirds of the body weight is composed of water; the proportion of intracellular and extracellular fluids is five-sevenths and two-sevenths.[12]

The problem of main interest here concerns reabsorption of fluid by osmosis and transudation from epithelial surfaces by hydrostatic pressure, aided by cell activity.

HYDROSTATIC AND OSMOTIC PRESSURES IN THE NOSE AND RESPIRATORY TRACT

The delicate epithelium lining the air passages, whether olfactory, ciliated or stratified, is capable of allowing water to pass more freely than through skin.



A B

Fig. 6.—A, olfactory recess of a normal carp (cyprinus carpio). There are scattered goblet cells in the skin (a.) and numerous goblet cells in the lamellae of the olfactory recess (b.). A, skin; B, olfactory recess of a carp (cyprinus carpio) after immersion for 2 hours in 2 per cent saline solution. The goblet cells in the lamellae are numerous and many are being discharged.

The relative thickness of the mucosa varies with the locality, being much thicker in the olfactory area; in a rabbit, for example, the relative measurements are 80 μ for olfactory, 43 μ ciliated, and 7 to 10.8 μ stratified epithelium (Fig. 1).

It is probable that the permeability is in the reverse order, the thin mucosa over the maxilloturbinal being two cells in thickness, with consequent freedom for passage of moisture.

Another factor of importance is the capillary blood supply, which in the stratified and ciliated membranes is copious, with large submucosal blood spaces or sinusoids; the olfactory region is much less vascular and does not respond to adrenalin, histamine or sympathetic control in the same active manner as the mucosa of the air conditioning area.

An obvious pervious spot is the olfactory recess of fish, which is always filled with water; in them and in mammals, with a film of moisture constantly covering the mucosal surfaces, problems both of hydrostatic pressure and of osmosis are entailed (Fig. 6).

Tissue fluid has a hydrostatic pressure of only 20 mm H_2O, but even this level is sufficient to cause transudation through the free epithelial surfaces, with subsequent evaporation into unsaturated inspired air (Fig. 2).

Since there is a temperature gradient in the nose, trachea, bronchi and bronchioles, at least in man, there will be evaporation of moisture on inspiration, with eventual saturation at body temperature before the alveoli are reached; there is condensation in the upper trachea and nose on expiration. This condensed water vapor, of negligible ionic content, will tend to pass into the epithelial cells and submucosa if its osmotic pressure exceeds the low hydrostatic pressure of the tissue fluids (Fig. 7).

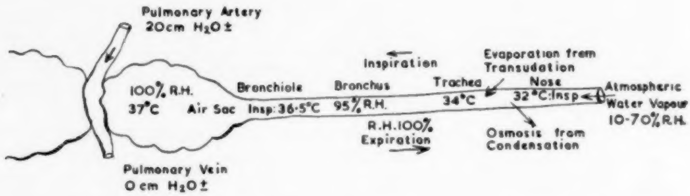


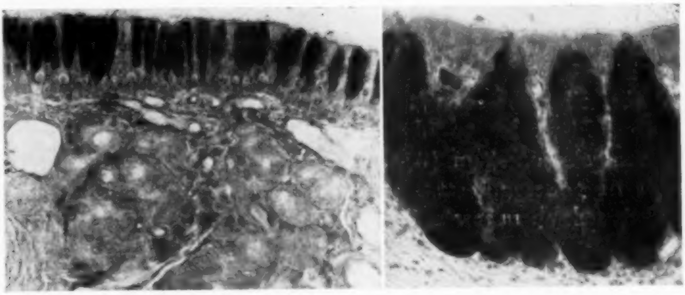
Fig. 7.—Temperature gradient in the respiratory tract. The figure represents the respiratory tract in Man from nose to air sac and illustrates the gradual rise of temperature on inspiration, with a corresponding drop on expiration. When air saturated with moisture at body temperature reaches the upper trachea and nose, the reduced temperature may lead to condensation, with passage of fluid into the tissues by osmosis.

SYMPATHETIC CONTROL

The normal regulator of the arterioles and capillaries of the nasal mucosa is the autonomic nervous system.

Hydrostatic pressure in the capillaries varies directly with the degree of dilatation of the arterioles, which sympathetic control maintains in a state of tonic contraction by constrictor impulses continually delivered to all vessels. Many capillaries are completely closed at rest and large numbers open up on necessity. Pressure in the capillaries, usually low, can be raised, often greatly.[16]

Increased blood supply, with increased transudation, allows a greater supply of heat and moisture to be delivered to inspired air for the purposes of olfaction, ciliary action and respiratory exchanges.



A B

Fig. 8.—A. Section through ciliated columnar epithelium of a dog (canis familiaris) showing numerous goblet cells containing mucus, stained with mucicarmine. There are also large subepithelial multicellular glands of serous type.

B. Section through ciliated columnar epithelium in the nose of a Pigeon (columba), to show the large multicellular mucous glands stained with mucicarmine.

Absence of sympathetic control, as in Horner's syndrome, leads to engorgement of vascular spaces in the nose; there is parasympathetic vasodilator innervation of the vessels.[15,19]

INFLUENCE OF CHANGES IN REACTION OF THE TISSUE FLUIDS

Any considerable rise above the normal pH 7.0 results in increased permeability of cell walls, with consequent swelling of tissue cells; increased transudation through the free epithelial walls will lead to escape of fluid, which will remain on the surface in excessive amount if not carried off by evaporation.

Such an occurrence is abnormal and is characteristic of allergic states; it is of interest to note the accuracy of reaction essential for normal function.

THE ROLE OF CALCIUM

Various examples could be given to illustrate the importance of calcium as an adjunct to impermeability; the essential presence of this element in cell membranes has already been mentioned.

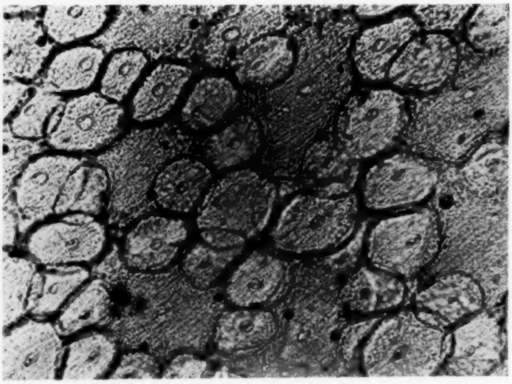


Fig. 9.—Skin from the belly of a Xenopus toad to show the numerous mucous glands and their ducts. The pigment cells are contracted as a result of the removal of the pituitary gland.

A marine worm, *Nereis diversicolor*, if placed in 20 per cent sea water swells up and then recovers; if in dilute sea water free of calcium it remains swollen.[14]

Another worm, Gunda, behaves somewhat similarly.

Calcium is essential, not only in cell membranes, but also in intercellular cement.[8] It is significant that the giving of calcium, preferably combined with acids, is of benefit to allergic subjects, not only in reducing transudation but also in controlling allergic edema of the tissues.

The intercellular spaces which are the main site of fluid exchange are filled with intercellular cement; this, presumably, is composed of calcium proteinate, a substance which can be washed away if the calcium content is deficient.[20] There is a reserve of calcium in the bones, but it is obviously not always available.

SECRETORY GLANDS

Goblet cells and numerous racemose glands produce mucus, a viscous fluid consisting of granules of insoluble mucin mixed with water; in certain mucosal regions there are serous glands, but many of these seem to produce a sero-mucinous secretion (Figs. 8, 10).

Although mucous glands are always present in the ciliated areas of the nose and respiratory tract, yet they are also found in the non-ciliated olfactory mucosa of cats; mucous glands are an essential constituent of gastric and intestinal mucosa, where they provide a mechanical protective covering.

The quantity of fluid that can be poured out by the nose makes it appear improbable that all could be derived from secretory glands, especially when the viscid nature of mucus is considered; transudation is essential. It was found by Florey, Carleton and Wells[9] that atropine led to an almost complete disappearance of mucus in the tracheal glands, but goblet cells were not appreciably affected; they found also that if dry air were passed over tracheal pouches of cats and dogs, after giving large doses of atropine, the amount of water given off was not reduced. They concluded that the bulk of water collected was the product of vascular transudation rather than of secretion from glands.

THE ROLE OF MUCUS

Mucus is produced by goblet cells in the olfactory recess of some fish, but not for moistening nor for ciliary action; the object appears to be waterproofing of a semipermeable membrane with a viscous layer containing insoluble mucin.

Mucus is present in quantity on the skin of fish, notably in eels and also in aquatic amphibia such as Xenopus toads, with the protective function of preventing passage of water by osmotic pressure; marine species are in danger of dessication and fresh water animals are liable to flooding (Fig. 10).

An eel with mucus removed dies in fresh water, and a toad similarly swells up if outpouring from its kidneys is prevented (Baldwin).[1] Outpouring of mucus is increased if a carp is placed in hypertonic saline (Fig. 6).

A covering of mucus is present in the nose and sinuses, in the trachea and bronchi, and in the stomach and intestines; the only common factor to ascribe as a function is assistance in the control of fluid exchanges, either by hydrostatic or by osmotic pressure.

A very remarkable observation has been made regarding the interrelation of skin and mucous membranes.

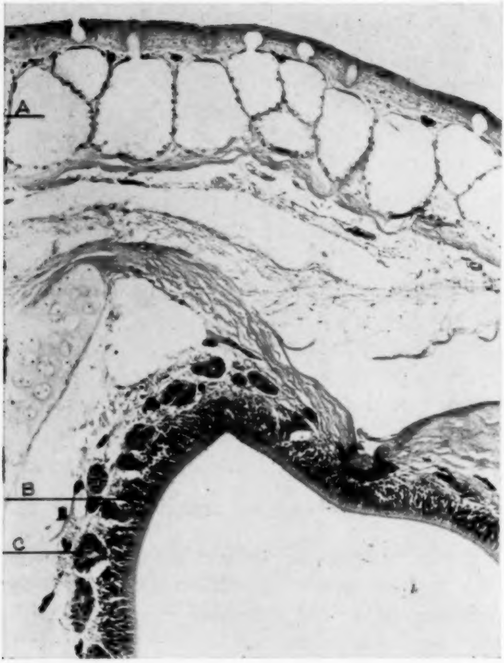


Fig. 10.—Section through the skin and nasal fossa of a Xenopus toad to show the big multicellular mucous glands beneath the skin. The pituitary gland had been removed to shrink the pigment cells or melanophores. A, subepithelial mucous glands; B, olfactory epithelium; C. Bowman's glands.

Fell has reported experiments, carried out in collaboration with Mellanby, on the effect of excess of Vitamin A on developing chick ectoderm.[5,6] Normally this tissue will grow into a keratinizing squamous epithelium, to act as a resistant barrier on the surface of the body; under the influence of Vitamin A there is, in place of keratinizing epithelium, columnar epithelium resembling nasal mucosa, with cilia which show activity and with numerous large goblet cells, producing mucus.

Vitamin A retards keratinization and its direct effects are seen in these experiments; the manufacuring plant of the cell, determined by the amount of vitamin A, seems to have the potentiality of producing either keratinized epithelium or cilia and mucous glands, the

mucus being produced in separate goblet cells and not in the ciliated epithelial cells.

The alternative holds good that absence of fat soluble vitamin A leads to excessive keratinization of the skin[4]; administration of the vitamin would, therefore, appear desirable to maintain the health of the columnar ciliated epithelium.

These observations help to throw light on the alternative means of protection of the body. Some species have a thick, keratinized skin, impervious to water, while others are provided with chitin; many fish, such as carp, have a thick but flexible skin studded with goblet cells (Fig. 6), and in the aquatic Xenopus toad the skin is thinner, with an underlying continuous layer of multicellular mucous glands (Figs. 9, 10).

The olfactory recess of some fish and the lining membrane of the ectodermal respiratory and alimentary tracts do not have a thick and impervious lining, since this would prevent not only olfaction, but also activity in transuding moisture or in absorbing food or water; it would also make ciliary action impossible.

There is, instead, a delicate epithelium, designed to carry on various functions, with its surface protected by a waterproofing layer of diluted mucus (Figs. 4 and 6).

In addition to exchange of fluid, secondary advantages of this covering are directed to ciliary action or to elimination of CO_2, all of which can continue uninterruptedly beneath the mobile and ever-changing protective blanket; the provision of an impervious epithelium would naturally eliminate ciliary action, intestinal absorption and respiratory exchanges.

It is important that the olfactory region should not be covered with a mucous blanket, since this might impede the passage of olfactory molecules.

As regards the association of mucus and ciliary action, it must be noted that ciliary streams flow more freely in dilute mucus, as occurs in allergic states.[18] Cilia carry out many functions—some of primitive origin, for instance, the propulsion of protozoa, or the nutrition and respiration of molluscs, which are carried on in a watery medium devoid of mucus. To mucus has been ascribed the function of pro-

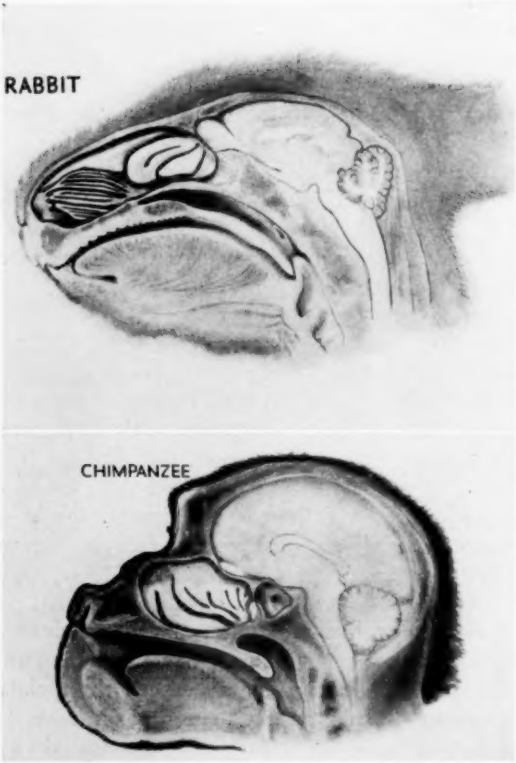


Fig. 11.—The nose of the rabbit (Lepus cuniculus) has a branching maxilloturbinal of very extensive area and an elaborate ethmoturbinal system with several endo- and ectoturbinals.

The chimpanzee (Anthropopithecus troglodytes) has a small flat and single scrolled maxilloturbinal and a single flat ethmoturbinal, furrowed in the position of previous subdivisions; there are no ectoturbinals and the sense of smell is feeble.

tecting the body against invasion by bacteria, by two methods. The first is an active bacteriostatic or bacteriocidal action formulated by Fleming, who ascribed this destruction to the presence of lysozymes.[7] In 1895 St. Clair Thomson and Hewlett examined cultures from the human nasopharynx after certain organisms had passed through the nose and came to the conclusion that there was a bacteriocidal process at work.[17]

The observations of others have, however, belittled the importance of lysozymes, considering them to inhibit nonpathogenic bacteria alone.

The second method of protection is mechanical, by removal of bacteria entangled in the blanket of mucus which covers epithelial surfaces in the nose and tracheobronchial tract; this blanket is propelled by cilia and the organisms are finally destroyed in the stomach by acid secretions.

Mucus is also said to have a digestive effect on pollens (Strömme).

Protection against virus infection also is ascribed as a function of ciliary activity.

A layer of secretion composed of diluted mucus is present on a considerable area of the nasal epithelium; in all species the olfactory area is devoid of cilia and consequently there could be no removal here of bacteria by streaming. There are no cilia on certain parts of the nasal septum, nor on the maxilloturbinal of some animals such as rabbits.

Cilia and a mucoid blanket are present in the trachea and bronchi of mammals but not in the atria or air sacs.

Mucus covers the epithelium of the stomach and small intestine and is considered by Florey to act as a protection against bacterial invasion; but here peristalsis and the passage of food and water propels and removes the debris in the absence of ciliary action.

There is no doubt that mucus is an important agent in protecting epithelial surfaces against bacterial invasion by inhibitory and scouring processes, but on the evidence available, including that of the olfactory recess of fish, this does not seem to be the only reason for its presence, nor that of most importance. The function of providing a resistant layer to the passage of water must be considerde to be one of considerable significance.

CONCLUSIONS

Having considered the various factors concerned in fluid exchanges it now remains to discuss the mechanism as it affects the nose and respiratory tract.

The same type of process that leads to outpouring of plasma from the capillaries, without the large protein molecules, appears to take place at the free surface of the epithelial cells bordering on the lumen of the nose, the trachea and the bronchi.

Under the influence of hydrostatic pressure in the tissue fluid, there is transudation, regulated by the sympathetic and parasympathetic systems, according to the necessities of inspired air for humidification up to saturation point (Fig. 2).

Insensible evaporation from the skin is a means of getting rid of much body heat by loss of water, independently of sweating; the fluid so lost has no ionic content.

Transudation through epithelial surfaces in the nose and respiratory tract appears to follow the same principle, with escape of moisture devoid of salts; return of any part of this fluid into the tissues by osmosis is improbable, since the overlying blanket of mucus takes up any moisture available and itself exerts slight osmotic pressure owing to the presence of salts.

How the autonomic nervous system controls the exact level of capillary constriction or dilatation and the emptying or filling of submucosal sinusoids, is not clear, but it would appear difficult for an accurate balance to be maintained between the fluid poured onto the epithelial surface and that removed by evaporation into the unsaturated inspiratory air stream.

It is suggested that in the absence of resistance by osmotic pressure, such as occurs in the air sacs and alveoli and between the tissue fluids and the capillaries, a partly impervious layer of diluted mucus is provided as a covering to the mucosal surfaces.

The particles of mucus are insoluble in plain water and their presence in the fluid covering will impede the passage of water into or out of the epithelial surface; fluid tending to escape from the surface of the mucous membrane must first dilute the layer of mucus to a degree where evaporation into the air stream becomes possible;

particular regard must be directed to the maintenance of the optimum degree of viscosity of the mucus for ciliary action.

This waterproofing layer would have the function of delaying and regulating escape of fluid, thus preventing flooding of the nose and air passages.

The distribution of mucus-secreting goblet cells and glands fits in with this proposal; such glands are absent in the olfactory mucosa, but the thickness of the epithelium and the paucity of underlying vascular spaces are reasons why the olfactory area plays little or no part in humidification (Fig. 1).

The olfactory region has its special function provided for. Again, in the terminal air sacs and alveoli there is no mucus, which would interfere with gaseous exchanges; but here, the force of osmotic pressure, opposing the low hydrostatic pressure, prevents excessive escape of fluid.

The provision of a layer of moisture in the nose and respiratory tract is essential for the functions of olfaction, respiratory exchanges and ciliary action; for the supply of fluid, evidence has been advanced in this communication in favor of transudation through the delicate permeable epithelium, under the influence of hydrostatic pressure, as being the main bodily source of supply; secretion from goblet cells and multicellular glands appears to play a minor part in humidification, but water vapor in inspired air is an important but variable factor.

The structure of the cell membranes is, of course, of great importance, both in transudation and in osmosis.

Osmosis is considered to come into play when water vapor condenses during expiration on areas of the trachea and nose at a temperature below that of the lower air passages; the moisture so deposited is practically distilled water (Fig. 7).

Calcium is known to be an essential constituent of cell membranes and of intercellular cement; its supply in adequate amount is essential, together with maintenance of the correct ionic content of tissue fluids. Deviations of either factor, together with sympathetic imbalance, result in abnormal permeability and excessive transudation or osmosis, as in allergic states.

It is suggested that the abnormal increase of goblet cells in some asthmatic subjects, with excessive secretion of viscid and obstructing mucus, may be a misdirected attempt at reduction of abnormal permeability of the epithelium. The permeability of the capillary endothelium is markedly increased by histamine, which has been shown by Dale to be released in allergic reactions.[3,11]

It is my belief, based on the evidence of comparative anatomy, but without experimental proof, that the problem of fluid exchanges is simpler in macrosmatic species[13] (Fig. 11A). In these animals there is an efficient air-conditioning apparatus provided by an elaborately branching maxilloturbinal body, which, on anatomical grounds, would appear capable of raising inspired air up to body temperature and of saturating it with moisture. Consequently there would be no temperature gradient in the respiratory tract and no necessity either for transudation or for osmosis in the lower respiratory passages; inspired air would reach the air sacs saturated with moisture and on expiration there would be no condensation, except perhaps in the anterior region of the nose. The blanket of mucus would thus insulate the epithelial surfaces, allowing ciliary action to continue unimpeded.

In microsmatic species, such as higher apes and man, regression of the air-conditioning system, with contraction of the mucosal area, is associated with diminution of the olfactory sense (Fig. 11B). The nose of these species is unable to bring inspired air up to body temperature and consequently moisture must be taken up from the trachea and bronchi until saturation is reached in the air sacs; some of this moisture condenses in the upper respiratory tract on expiration. The problems connected with transudation under hydrostatic pressure and those of osmosis are consequently more obvious when the nasal air conditioning mechanism is not highly efficient (Fig. 7).

As regards other functions, the mucous blanket, propelled by ciliary action, is of considerable importance in the prevention of bacterial invasion; but it is here suggested that it is not the only function, nor that of most vital significance.

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I wish to express my indebtedness to Professor Slome for his kindness in reading this and other contributions. His comments have been most valuable to me in this complicated physiological subject.

Once again I acknowledge with gratitude the facilities extended to me at the Royal College of Surgeons of England and at the Ferens Institute of the Middlesex Hospital.

I have also had the benefit of Professor Dohlman's critical opinion.

REFERENCES

1. Baldwin, E.: Introduction to Comparative Biochemistry. Cambridge Univ. Press, 1948.

2. Causey, G.: Personal Communication, 1957.

3. Dale, H., and Laidlaw, J.: Jour. Physiol. 52, 1919.
Josiah Macy Jr. Found., N. Y., 1953.

4. Evans, C. Lovatt: Principles of Human Physiology. London, Churchill, 1956.

5. Fell, Honor B.: Connective Tissues. Trans. 4th Conference. Edit. C. Ragan. Josiah May Jr. Foundation, New York, 1953.

6. Fell, Honor B., and Mellanby, E.: Jour. Physiol. 119:470, 1953.

7. Fleming, A.: Jour. Laryngol. and Otol. 43:385, 1928.

8. Florey, H.: Lectures on General Pathology. London, Lloyd-Luke, 1954.

9. Florey, H., Carleton, H. M., and Wells, A. Q.: British Jour. Exper. Pathol. 13:269, 1932.

10. Gamble, J. L.: Lane Medical Lectures, Stanford Univ. Pub. 5:1, 1951.

11. Lewis, T.: Blood Vessels of the Human Skin and Their Responses. London, Shaw, 1927.

12. Marriott, H. L.: Ann. Royal College Surg., England, 7:339, 1950.

13. Negus, V. E.: Brit. Med. Jour. 1:367, 1956.

14. Prosser, C. Ladd: Comparative Animal Physiology. Philadelphia and London, B. Saunders and Co., 1950.

15. Slome, D.: Scientific Basis of Medicine. Series of Lectures, Athlone Press (in the press).

16. Starling, E. H.: Principles of Human Physiology. Ed. C. Lovatt Evans, London, Churchill, 1941.

17. Thomson, St. Clair, and Hewlett, R. T.: Med. Clin. Trans. 78:239, 1895.

18. Tremble, G. E.: Laryngoscope 58:206, 1948.

19. Tschallusow, M. A.: Pflug. Arch. des Physiol. 151:523, 1913.

20. Zweifach, B. W.: Connective Tissues. Trans. 5th Conference. Edit. C. Ragan. Josiah Macy Jr. Foundation, New York, 1954.

XXV

TRANSORAL INTRALARYNGEAL APPROACH FOR ARYTENOIDECTOMY IN BILATERAL VOCAL CORD PARALYSIS WITH INADEQUATE AIRWAY

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Vocal cord paralysis may result from a variety of causes. It may be produced by either a central or peripheral lesion. The paralysis may be seen in peripheral neuritis secondary to acute infectious diseases, diphtheria, typhoid fever or syphilis. It may be seen as a complication of bulbar poliomyelitis. The condition has also been reported in various drug poisonings. In rare instances it has occurred as a complication of acromegaly. A mediastinal tumor or enlarged inflammatory or metastatic lymph node, enlarged thyroid gland, or direct involvement by malignancy of the thyroid gland may involve the recurrent laryngeal nerve and produce this complication. Crico-arytenoid fixation may occur in blastomycosis or in nonspecific infections and cause a fixation of the vocal cords. The most frequent cause of bilateral vocal cord paralysis seen by the author with a resulting inadequate laryngeal airway has occurred from injury to the recurrent laryngeal nerves during the process of removal of the thyroid gland.

A paralyzed vocal cord may assume a position at or near the median line or a more lateral or so-called paramedian position. In a unilateral vocal cord paralysis, if the vocal cord remains in the median line, there is usually no hoarseness because the opposite normal functioning vocal cord may then approximate the paralyzed cord on phonation and produce a normal voice. If the unilateral paralyzed vocal cord is in the lateral position, hoarseness will be present. Unilateral vocal cord paralysis usually presents no serious problem, since the excursion of the opposite normal vocal cord is sufficient for an adequate airway, but a few rare cases have been observed where an

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Read at the Sixth International Congress of Otolaryngology, Washington, D. C., May 1957.

inadequate airway has been present. If a bilateral vocal cord paralysis or fixation occurs with the vocal cords assuming a median line position, the patient is placed in an extremely critical condition. In this instance an emergency tracheotomy may be necessary to establish an adequate airway. In some individual cases, patients have been observed with a small glottic opening and have become adjusted to a restricted airway, developing dypsnea and stridorous breathing on exertion, or associated with acute respiratory infections which make it then advisable to proceed with tracheotomy. An occasional patient may be observed with paralyzed vocal cords at different levels so that the glottic opening, although apparently constricted, allows for normal respirations in limited activities.

In patients with an inadequate airway due to vocal cord paralysis or crico-arytenoid fixation, and who have shown no improvement after six months from the time of onset of this complication, one should consider treatment to improve the airway. Only on very rare occasions may the patient prefer to retain a tracheotomy tube, or to use a valve tube as described by New or Tucker. Many surgical procedures have been presented for the treatment of this condition, but time does not allow the review of these various operations. The following surgical procedure has been successfully applied by the author with excellent end results.

TRANSORAL INTRALARYNGEAL APPROACH FOR ARYTENOIDECTOMY[1]

If the patient has not had a previous tracheotomy, the first step is to carry out this procedure under local anesthesia. It should be kept in mind that as the result of an inadequate airway, preoperative sedation should not be administered. The tracheotomy can be done immediately preceding the arytenoidectomy or one week before, according to the general physical condition of the patient.

Following the tracheotomy, the oropharynx, hypopharynx and larynx is anesthetized by the application of a topical anesthetic of the surgeon's choice. General anesthesia is then induced by the use of pentothal sodium administered intravenously (to which curare or curare-like substances may be added), after which the suspension laryngoscope is placed in position. Although other types of self-retaining laryngoscopes have been used in carrying out this operation, the Lynch suspension apparatus affords the best visualization and the widest field for using several instruments in the larynx at one time. After adequate exposure of the larynx has been obtained,

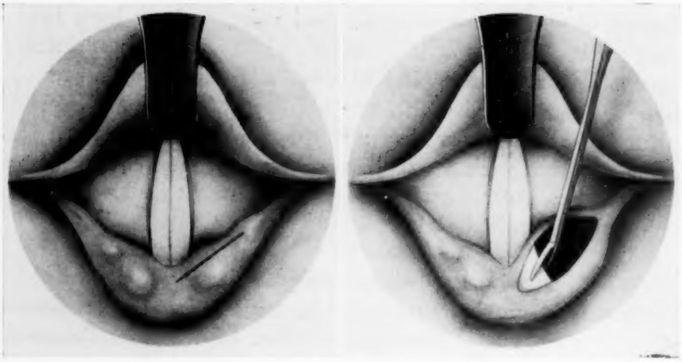

Fig. 1.—The incision is made in the aryepiglottic fold 1 to 2 mm medial to the smaller cartilages of Wrisberg and Santorini.

Fig. 2.—The cartilage is removed with the laryngeal forceps after all the muscle fibers have been separated from the cartilage.

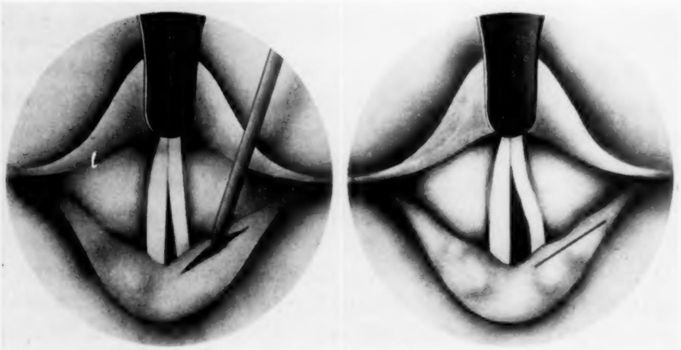

Fig. 3.—Electrocoagulation is carried out.

Fig. 4.—The vocal cord is displaced in a lateral position.

Fig. 5.—Acrylic mold which is inserted between the vocal cords.

further applications of the topical anesthetic are then made to the vocal cords and the aryepiglottic folds. An injection of procaine with epinephrine, or epinephrine with saline solution may be made into the aryepiglottic fold to minimize bleeding. An incision 1 to 1½ centimeters in length is made over the superior surface of the arytenois cartilage beginning posteriorly and extending in an anterolateral direction in the aryepiglottic fold, approximately 1 to 2 millimeters medial to the cartilages of Santorini and Wrisberg, so as to avoid these smaller cartilages in the dissection (Fig. 1). The original incision is carried deep into the tissues, down to the arytenoid cartilage. An alligator type of laryngeal grasping forceps is passed deep and lateral to the cartilage. This forceps slips readily along the side of the arytenoid cartilage because the muscle fibers on the lateral surface of this cartilage are loosely attached. On removing the forceps from this area, the forceps is opening detaching these muscles from the cartilage. The superior border of the arytenoid cartilage is better identified by carefully dissecting the mucous membrane from the medial aspect of the cartilage, and then the muscle attachments are separated on this side of the cartilage with a sharpened Lynch elevator. The mucous membrane in this area is close to the cartilage. The muscle fibers on this surface are more firmly attached. On dissecting the muscle attachments from the cartilage on the medial aspect, it is important to avoid tearing or masceration of the mucous membrane, because scarring in this area which can follow may nullify one's attempt to increase the glottic opening. Following removal of the muscle attachments from the cartilage, the cartilage itself is disarticulated and then removed (Fig. 2). It is very important to remove all portions of this cartilage. It is not unusual to have fragmentation of

this cartilage occur during the dissection. Following removal of the cartilage, a guarded curved electrocautery point may be deeply inserted through the incision lateral to the posterior two-thirds of the vocal cord and mild electrocoagulation applied (Fig. 3). Electrocoagulation is then carried out on the lateral surface of the wound. Lateral fixation of the posterior two-thirds of the vocal cord may be made more certain by the additional scarring which follows the use of the electrocoagulation (Fig. 4). Bleeding throughout the course of the operation is usually quite minimal and can be controlled with the use of topical epinephrine applied with a cotton applicator. Following electrocoagulation, an acrylic obturator may be introduced between the vocal cords (Fig. 5). This is anchored below to the flange on the tracheal cannula, and above through the nasal passage to the cheek. The obturator is left in place for approximately six or seven days. This helps to approximate the wound edges and eliminates the need for a suture.

The patient is instructed not to talk during the period of time that the acrylic obturator is in position between the vocal cords. The patient is placed on intravenous fluids for 24 to 48 hours following surgery before being started on liquids. The tracheal cannula may be occluded within ten days to six weeks. If the patient is able to tolerate occlusion of the tracheal cannula for one week, and if in the mind of the observer the glottic opening is adequate, the tracheal cannula may then be removed. A sterile dressing is placed over the tracheal stoma, and in 7 to 14 days this opening into the treachea will close.

COMMENT

There have been continued reports of successful results by many surgeons using the transoral intralaryngeal approach in over 300 cases. The author has had successful end results in 55 out of 56 cases. In 7 cases it was necessary to proceed with arytenoidectomy on the opposite side to produce a more adequate airway.

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REFERENCE

1. Thornell, W. C.: Intralaryngeal Arytenoidectomy in Bilateral Abductor Vocal Cord Paralysis. S. G. and O. 95:63-66, 1952.

XXVI

TRENDS IN RADICAL FRONTAL SINUS SURGERY

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The purpose of this paper is to comment on the changes in the management of frontal sinusitis which have occurred as a result of: 1) antibiotic control of bacterial infection, 2) a better understanding of the physiology of the nose and accessory sinuses and 3) improvements in techniques and training.

ANTIBIOTICS

These drugs are to be considered as specific rather than in the popular broad spectrum sense. At the Massachusetts Eye and Ear Infirmary we have facilities for early bacteriological identification including tests for sensitivity to antibiotic drugs. Also we have that most important factor, enthusiastic support from the bacteriological department. But although it is thus possible to start the drug of choice early in the disease, I wish to emphasize that these drugs are to be used in conjunction with other established procedures. Antibiotic treatment, where normal drainage from the sinus is established, has shifted many cases previously considered amenable only to radical surgery to the more conservative procedures. Also, as certain of the older techniques failed because of our inabality to control the infection in the postoperative period, we may now reconsider them, thanks to these drugs. This factor has a direct bearing also on those cases of osteomyelitis formerly considered as surgical emergencies. When effectively given, antibiotics grant us respite in time to evaluate the severity of the disease and to exercise more critical judgment. We need not be stampeded by our former fears into an early frontal ostectomy. Also it is now practical to consider the normal physiology of the nose and sinus due to the respite afforded by the drugs.

Read at the Sixth International Congress of Otolaryngology, Washington, D. C., May 1957.

PHYSIOLOGY OF THE NOSE AND SINUSES

One can see here the effect on our thinking of the studies of Hilding,[1] Proetz[2] and others on the physiology of the nasal mucous membrane with emphasis on preserving the function of the cilia in maintaining the normal flow of secretion from the sinus into the nose. Also the advice of Mosher[3] to respect the virginity of the nasofrontal duct gains greater significance, for trauma to this duct leads to stricture. Walsh[4] and Brown[5] who have modified the Jansen Lynch operation and Bergara,[6-8] Tato,[9] Gibson and Walker,[10] and Macbeth,[11] in the anterior osteoplastic flap approach, all respect the cilia of the nasofrontal duct because of their physiological importance.

TECHNICAL DEVELOPMENTS

In regard to technical developments in the broad sense to include the training of the specialist, the author wishes to mention without extended comment the following:

a. *Education.* Especially to be cited as contributing to our general advance is the progress made in the following areas:

1. The increase of postgraduate courses and residencies in our specialty.

2. The establishment of specialty boards which have raised the level of proficiency by requiring postgraduate training as a qualification for certification.

3. The general spread of knowledge through the translation and abstraction of foreign language literature.

4. The increased interest in national and international society proceedings.

b. *Roentgenology.* X-ray diagnosis based on improved techniques has greatly aided the rhinologist in determining which operation is indicated for a specific case.

c. *Reconstructive Surgery.* The plastic surgeon can now repair the damage of a disfiguring operation so that this one great objection to the total obliteration technique of Riedel[12] is removed.

d. *Mechanical Improvements.* The general improvements in instruments and especially in the electrically operated drills, burrs, and fine circular saws make it possible to perform more precise operations.

e. *Tissue Banks.* These banks which are now in existence in many of the large general hospitals provide bone, fat, or fibroblastic tissue which can be used on call for a reparative procedure. This is of great help when it is impractical to use autogenous grafts.

f. *Anesthesiology.* Advances in anesthesia, notably the use of the newer basal drugs and intravenous anesthetics, the safety afforded by the endotracheal tube and the better techniques in nerve block, give the surgeon a much better chance to accomplish his task more effectively.

Bearing these points in mind, let us proceed to the thesis of this paper—The Trends in Surgery of the Frontal Sinus.

Operative techniques for relief of frontal sinus can be classified in two groups according to their objectives, which are determined by the clinicopathological picture.

The objectives in the first group where there is still a possibility of preserving normal function are: a) to resolve or eradicate the pathology within the sinus; b) to preserve or obtain surgically a patent nasofrontal duct.

The objective in the second group where the pathology or clinical picture is such as to preclude a return to normal physiology or where the likelihood of severe reinfection is very great, is the total obliteration of the frontal sinus and a permanent occlusion of the nasofrontal duct.

GROUP I

Cases Where the Preservation of Function Is Possible. Prior to chemotherapeutic and antibiotic drugs, the means at the disposal of the surgeon were limited in the acute cases to local shrinking of the mucous membrane to relieve intranasal obstruction, drugs for the control of pain and the application of external heat. Minor surgical procedures such as turbinotomy, anterior ethmoidectomy or submucous resection were indicated in those patients who failed to respond within a reasonable time to these conservative measures. One other procedure which has come down to us from those days with increased favor is the early trephination for acute frontal sinus empyema as pointed out by Boies.[13] In all our enthusiasm for recent antibiotic developments, let us not forget that these conservative procedures are still indispensable in our armamentarium.

In more chronic cases the techniques range from those just mentioned to more extensive procedures. The earlier surgeons tried to enlarge the nasofrontal drainage by combined intranasal anterior ethmoidectomy, removal of the agger nasi cell and the superior nasal spine. To maintain this patency various devices were employed, such as the Ingal's[14] gold tube or the mucoperiosteal flap of Halle.[15] To go a step further, where tissue within the sinus was so chronically infected that its removal was necessary, the external approach was developed. To simplify the historical discussion of these techniques, let us call attention to two points of divergence in concept.

1) The external fronto-ethmoidectomy with destruction of the anatomical nasofrontal duct as a result of the ethmoid exenteration.

2) The external frontal sinusotomy with preservation of the nasofrontal duct.

The first trend is typified by the Jansen Lynch operation in the United States. This is evolved from the Ogsten[16]-Luc[17]-Jansen[18] techniques. The salient points of the operation as described by Lynch[19] are as follows:

1. Killian incision with elevation of the periosteum from the roof of the orbit, lacrimal bone and lamina papyracea.

2. Careful separation of the lacrimal sac and the pulley.

3. Complete removal of the bone of the floor of the sinus to the outermost limit.

4. Curettage of all sinus membrane to bare bone.

5. Retraction of lower part of incision to inspect deeper regions.

6. Removal of lacrimal bone and lamina papyracea to within a few millimeters of optic foramen.

7. Resection of the nasal process of the superior maxilla.

8. Entire ethmoidectomy including removal of middle turbinate.

9. Removal of anterior sphenoid wall.

10. Nasal spine of the frontal bone not interfered with, wound

closed without drains externally, rubber tube and gauze drain, intranasally.

This operation has had its failures as well as its successes as pointed out by Anderson[20] in 1932. With the same objective, the author[21] studied the cases at the Massachusetts Eye and Ear Infirmary from the years 1933 to 1942. This was just before the antibiotic era and during the early days of chemotherapy. In a series of 117 Lynch operations, 34 required reoperation because of: 1) obstruction of the nasofrontal duct by scar tissue; 2) incomplete removal of the sinus membrane and incomplete ethmoidectomy. Weille[22] in 1946 extended this study at the Massachusetts Eye and Ear Infirmary. His conclusions also point up the problem of nasofrontal duct occlusion.

To maintain the nasofrontal passage where the anatomical duct is removed in the course of the ethmoidectomy, the following techniques have been employed:

1. Skin grafting of the duct with temporary inlying tube to hold the graft in place while epithelialization takes place, as described by Hoople.[23]

2. Mucous membrane flap to line the new nasofrontal duct according to the techniques of Sewall[24] and Boyden[25] and Mithoefer.[26]

3. Temporary acrylic mold obturators of Erich and New.[27]

4. Polythylene or tantalum tubes or foil inlays to prevent adhesions according to the techniques of Goodale,[28] Brown,[5] Weille.[22]

These devices have helped maintain patency between the nose and sinus but have not done away with the problem of reinfection. However, there has been a notable improvement in that the scar tissue obstruction is avoided and even if reinfection occurs, drainage is maintained and by proper antibiotic treatment the severity of the case is more easily controlled. In selected cases the Lothrop[29,30] operation may be considered which removes the interfrontal septum and the upper portion of the nasal septum and the nasal spine through an external approach thus providing a very large passage into the nose.

The second trend follows a different concept. Here the nasofrontal duct is preserved as it is believed that the untraumatized natural epithelium in this region is the best guarantee for subsequent

normal drainage. This follows the principles of Mosher[3] and is backed up by the studies of Hilding,[1] Walsh,[4] Proetz,[2] and others on the physiology of nasal mucous membrane.

There are two significant techniques:

1. Walsh[4] advocates an approach through the sinus floor, removal of the floor and the diseased mucous membrane of the sinus but only down to the vicinity of the duct. A small portion of the floor with intact mucous membrane is left undisturbed just a few millimeters above the nasofrontal duct.

2. The osteoplastic approach with temporary reflexion of the anterior wall of the sinus. This approach gives the largest exposure of the sinus cavity and an unparalleled opportunity to evaluate the pathology. Also as one can visualize especially well the funnel leading to the nasofrontal duct, it is much easier to avoid damage to its mucosa if the surgeon decides not to enlarge it or if he must enter the anterior ethmoid, he can do so without damage to the orbital bony wall. This approach was first described by Brieger[31] and Schornborn,[32] later was advocated by others, notably Winkler,[33] Beck,[34] Hoffman,[35] and more recently by Bergara,[6-8] Tato,[9] Gibson and Walker,[10] Macbeth,[11] and Vadala and Somers.[36]

GROUP II

Cases Where Restoration of Function Is Not Possible. In cases where it is obvious that the disease is so chronic and irreversible that the idea of maintaining patency from the sinus into the nose is discarded, a complete obliteration of the frontal sinus cavity with occlusion of the nasofrontal duct is sought. The mucous membrane of the sinus, of course, has to be totally removed. These procedures were developed by Kuhnt,[37] Ropke,[38] Riedel[12] and Killian[39] and were intended to be definitive operations. The first three by removing the anterior wall of the sinus allowed the surgeon to inspect the cavity easily and directly and the removal of all infected membrane was thereby facilitated. Reinfection from the nose led to the extension of the technique to include a complete ethmoidectomy. The resulting obliteration of the nasofrontal passage by granulations and fibrous adhesions then sealed off the communication with the nose. The operation failed to find general favor because of the severe deformity and the inevitable development of mucoceles when one failed to remove thoroughly the mucous membrane. Even Killian's[39] operation for

preserving the supraorbital ridge did not fulfill the early hopes of its advocates because of mucoceles and sequestration of the bridge. The most radical obliteration advocated by Mosher,[3] the total removal of the anterior and posterior walls and closure of the nasofrontal duct by curettage, insured the prevention of mucoceles by removing the bony substructures on which lay the mucous membrane.

The original objections to the Riedel operation have been offset by the great advances in reconstructive surgery. The use of autogenous bone grafts taken from the iliac crest and shaped to the contours of the forehead have given beautiful results. In the author's[40] experience this operation or the one to be mentioned next become the techniques of choice in the severely chronic case.

Of recent years the osteoplastic anterior wall approach has received renewed attention in obliterative surgery. Tato[9] and Bergara[6-8] both make an osteoplastic flap to expose the sinus cavity. The former measures by means of a grille pattern taken from the x-ray films, the boundaries of the sinus. After exposing the periosteum of the frontal bone by elevation of the overlying tissues, he outlines his incision through the periosteum by means of this grille and then with a specially constructed electrically driven drill he enters the sinus and makes a semicircular cut through bone and periosteum from the medial limit of the sinus to the outer lateral limit following the contours of the sinus. The two limits are then further freed from the supraorbital rim by blows with a straight edge chisel. One can then pry up the flap and thus obtain a linear fracture through the thin bone of the floor. As the flap is turned downward, the entire cavity is revealed directly to inspection. All membrane is removed down to the nasofrontal duct at which point a cuff of mucous membrane is made and invaginated into the nose. The cavity is then filled by a graft of fascia lata, or abdominal aponeurosis with attached fat, or by blood clot. The bone flap is then replaced and the wound closed. Bergara enters the sinus by a series of small trephinations along a curved line just within the confines of the sinus, probing after each drill hole to determine the exact point to make the subsequent one. These drill holes are then connected and the flap is turned down in the same manner as just described. With some differences in technique both Tato and Bergara advocate the anterior osteoplastic approach and the filling of the cavity by tissue implant, blood clot, or gelatin foam.

Macbeth,[11] using the osteoplastic anterior wall approach, relies on the osteogenesis of the denuded bone and the obliteration of the naso-

frontal duct to produce a sclerosis of the cavity in all intents an obliteration. This follows the principle advocated by Woods,[41] who after removing all the mucous membrane of the sinus and the duct in a modified Jansen Lynch operation does not deem it necessary to maintain the patency of the duct.

REPORT OF CASES WITH ANTERIOR WALL APPROACH

Recently the author has operated upon six cases by means of the anterior osteoplastic flap. The technique of Tato was followed in general but the bone was cut with a bevel by means of a small very thin circular saw instead of by the drill. This allowed perfect reapproximation when the flap was returned to its original position. A very satisfactory view of the sinus cavity was obtained and in cases where total removal of the mucous membrane was desired not only could it be dissected away more easily but the underlying bone could be polished by an electric burr thereby removing all microscopic remnants of membrane.

One case of osteoma was approached in this fashion, and I believe the success in removing the tumor without damage to the dura was because of the ease of detaching the growth by mallet and chisel through this wide exposure.

A case of pyocele was easily approached in this manner. The medial half of the sinus being normal, it was merely necessary to remove the cyst which lay at the lateral extremity of the sinus. The rest of the cavity being lined by normal membrane was not touched. In the other four cases abdominal aponeurosis and fat were used to fill the cavity after occlusion of the duct in the manner of Tato. I would recommend this approach as a valuable addition to our techniques for the following reasons: 1) better visualization of the entire cavity; 2) easier and therefore more thorough removal of mucous membrane; 3) because of better visualization the nasofrontal duct can be better preserved in the cases where the cavity is to be maintained or in cases where obliteration is desired it can be easily closed; 4) the cosmetic result is excellent as the bony structure of the forehead is preserved.

COMMENT

1. There is a definite trend to conservatism in reversible cases with emphasis on antibiotic treatment and early external trephine in undraining acute cases.

2. A tendency to leave the nasofrontal duct intact in external operations where return to normal function is to be expected.

3. The rationale of the external combined fronto-ethmoid operation is open to question because of the likelihood of nasofrontal occlusion and the tendency to reinfection. The employment of plastic obturators, metal tubes or foil, and mucous membrane grafting would seem to imply that this technique must rely on accessory procedures to maintain an opening.

4. For definitive procedures in severe cases, total obliteration is gaining favor, either total ablation followed by bone graft repair, or osteoplastic temporary flap and tissue implant.

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REFERENCES

1. Hilding, A.: Four Physiological Defenses of the Upper Part of the Respiratory Tract: Ciliary Action, Exchange of Mucin, Regeneration and Adaptability. Ann. Int. Med. 6:227, 1932.

Ibid.: Physiology of Drainage of Nasal Mucus, Cilia, and Mucin in Mechanical Defense of Nasal Mucosa: motion picture demonstration. Annals of Otology, Rhinology and Laryngology 41:52, 1932.

Ibid.: Physiology of Drainage of Nasal Mucus: Flow of Mucus Currents through Drainage Systems of Nasal Mucosa and Its Relation to Ciliary Activity. Arch. of Otolaryngology 15:92, 1932.

2. Proetz, A. W.: Studies of Nasal Cilia in Living Mammal. Annals of Otology, Rhinology and Laryngology 42:778, 1933.

3. Mosher, H. P.: My Milestones. Laryngoscope 56:11 (Nov.), 12 (Dec.) 1946.

4. Walsh, T. E.: The Role of the Ostium and Nasofrontal Duct in Postoperative Healing. Laryngoscope 53:75 (Feb.) 1943, a. Personal Communications.

5. Brown, J. Mackenzie, and Goodhill, V.: Treatment of Acute Frontal Sinusitis (Preserv. of Naso-frontal Mucosa). Annals of Otology, Rhinology and Laryngology 64:291-299 (Mar.) 1955.

6. Bergara, A. R., and Itoiz, O. A.: Experimental Study of the Behavior of Adipose Tissue within the Frontal Sinus of Dog. Argent. Rev. Oto-Rhino-Laryng. 184-192 (July-Sept.) 1951.

7. Bergara, A. R., and Itoiz, O. A.: Present Status of the Surgical Treatment of Chronic Frontal Sinusitis. Arch. of Otolaryngology 61:616-628, 1955.

8. Bergara, A. C., and Bergara, C.: Chronic Fronto-ethmoidal Sinusitis. Osteoplastic Method According to Author's Technique. Annals of Oto-Rhino-Laryngology, Uruguay, 5:192-200, 1955.

9. Tato, J. M., Sibbald, D. W., and Bergaglio, O. E.: Surgical Treatment of the Frontal Sinus by the External Route. Laryngoscope 64:504-514 (June) 1954.

10. Gibson, T., and Walker, F. M.: Large Osteoma of the Frontal Sinus: A Method of Removal to Minimize Scarring and Prevent Deformity. Brit. J. Plast. Surg. 4:210 (Oct.) 1951.

11. Macbeth, R.: The Osteoplastic Operation for Chronic Infection of the Frontal Sinus. Jour. of Laryngol. and Otol. 68:7 (July) 1954.

12. Riedel: Schenke Inaug. Dissertation, Jena, 1898.

13. Boies, L. R.: Acute Frontal Sinusitis. The Trephine Operation for Drainage in Selected Cases. Laryngoscope 52:458:468 (June) 1942.

14. Ingals: New Operation and Instruments for Draining the Frontal Sinus. Laryngoscope, p. 644, 1905.

15. Halle: Die Intranasalen Operationen bei Eitrigen Erkrankungen der Nebenhöhlen der Nase. Archiv. f. Laryng. 1 Bd 29, H. 1, S. 73, 1914.

16. Ogsten: Trephining the Frontal Sinus. Med. Chronicle 1:235, 1884.

17. Luc: Lecons sur lè suppurative etc., p. 291, Paris, 1900.

18. Jansen: Neue Erfahrungen uber chronische Nebelhöhleneiterungen der Nase. Arch. f. ohrenhk, Bd 56, S. 110, 1902.

19. Lynch, R. C.: The Technique of a Radical Frontal Sinus Operation Which Has Given Me the Best Results. Laryngoscope 31:1:1-5, 1921.

20. Anderson, C. M.: External Operation on the Frontal Sinus—Causes of Failure. Arch. Otolaryng. 15:739-745 (May) 1932.

21. Goodale, R. L.: Some Causes for Failure in Frontal Sinus Surgery. Amer. O.R.L. 51:648 (Sept.) 1942.

22. Weille, F. L.: The Problem of Secondary Frontal Sinus Surgery. Tr. A.L.A. 67:150, 1946.

23. Hoople, G. O.: An Otolaryngologist's Experience in World War II. Am. O.R.L. 55:121 (Mar.) 1946.

24. Sewall, E. E.: External Fronto-Ethmo-Sphenoid Operations. Arch. of Otolaryng. 20:1:57-60 (July) 1934.

25. Boyden, G. L.: Surgical Treatment of Chronic Frontal Sinusitis. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 61:558 (June) 1952.

26. Mithoefer, W.: External Operation on the Frontal Sinus—A Critical Review. Arch. of Otolaryng. 7:133-149 (Feb.) 1928.

27. Erich, J. B., and New, G. B.: An Acrylic Obturator Employed in the Repair of an Obstructed Fronto-nasal Duct. Tr. Am. Academy of Ophth. and Otolaryng. 51:628-632 (July-Aug.) 1947.

28. Goodale, R. L.: The Use of Tantalum in Radical Frontal Sinus Surgery. Am. O.R.L. 54:747 (Dec.) 1945.

29. Lothrop, H. A.: Frontal Sinus Suppuration. Ann. of Surgery, pp. 937-957 (June) 1914.

30. Lothrop, O. A.: An Evaluation of the Lothrop Operation on the Frontal Sinus. Arch. Otolaryng. 33:73 (Jan.) 1941.

31. Brieger: Arch. J. Ohrenheilkunde, Vol. 39, p. 213.

32. Schörnborn: Wittkop Dissertation, Wurzburg, 1894.

33. Winkler: Beitrag zur osteoplastischen Freilegung des sinus Frontalis Verh. deutsch otol Gersellsch S 128, 1904.

34. Beck, J. C.: A New Method of External Frontal Sinus Operation without Deformity. J. A. M. A. (Aug. 8) 1908.

35. Hoffman: Osteoplastic Operations on the Frontal Sinuses for Chronic Suppuration. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, p. 598, 1904.

36. Vadala, A. J., and Somers, K.: Osteoma of the Frontal Sinus. Arch. Otolaryngology 50:618-633 (Nov.) 1949.

37. Kuhnt: Uber die entzündliche Erkrankungen d Stirnhöhlen etc. S. 207, 1895.

38. Ropke: Die Radikal operation bei Chronischen Eiterungen etc. Arch f Laryng. Bd 8, 1898.

39. Killian: Bemerkungen zur Radical Operation etc. Verh d Vereins suddeut Laryng s. 21, 1904.

40. Goodale, R. L.: The Radical Obliterative Frontal Sinus Operation—A Consideration of Technical Factors in Difficult Cases. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 64:470 (June) 1955.

41. Woods, R. R.: Proceedings of the Fourth International Congress of Otolaryngology, London, 1949, 1, 109.

XXVII

ANATOMICAL ETIOLOGICAL FACTORS IN CHRONIC MIDDLE EAR DISCHARGE

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Today's subject covers in fact the center of my interest for many years. For definite reasons, at the last Congress of Otolaryngology in Amsterdam I felt compelled to suggest the following: "I believe that we have now reached the moment where it is necessary to take problems of chronic otitis into renewed discussion, perhaps as the main problem of the next-coming Congress."

The fact that this has become a reality today, I do not for a moment suggest is the outcome of my proposition in 1953. To me, however, the choice of this main subject for the present Congress seems significant. It discloses, most conclusively, an increasingly common conviction that we have to re-examine generally held views on the so-called chronic otitis and, in a critical and scientific way, enter further into problems concerned. Otherwise we cannot proceed in diagnostic and prognostic skill, nor judge improvements achieved in modern therapeutical methods.

It is thus my sincere hope that this Congress may bring widened knowledge to all of us, brought forth by all contributors on this theme and by ensuing discussion.

THE PROBLEMS

My subject deals above all with the etiology of tympanic membrane perforations as related to the small mastoid air cell system. Indispensable for relevant conclusions seems to be a thorough knowledge of: 1) the anatomical size variation of the air cell system, 2) the

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anatomical or pathological nature of small air cell systems, and 3) the pathogenesis of central and marginal perforations.

MATERIALS AND METHOD OF INVESTIGATION

My data represent studies of a total of over 1,600 individuals:

1. 320 subjects, representative of the population, were collected for registration of the anatomical size variation of the mastoid air cell system.

2. An additional 1,039 individuals, representative of families, uniovular and binovular twins, served for registration of the part played in developmental growth by heredity and environment, respectively.

3. A final 275 patients with central and marginal tympanic membrane perforations served for registration of relationship between relevant size and perforation.

In the various groups both ears of all individuals were otoscopically and radiologically examined. The area of the mastoid air cell system on the x-ray film was measured with a planimeter.

Planimetry is quickly and easily performed. A film illustrating the x-ray projection as well as the planimetric method will be shown at the time reported in the program.

The advantage of measuring with a planimeter, allowing of numerical measurements and an exactness of ten square millimeters, as compared to the inexact ocular estimation of relevant size I was able to demonstrate at the Nordic Congress of Otolaryngology at Helsinki in 1954.

THE MASTOID AIR CELL SYSTEM

1. *The Air Cell.* As regards the single mastoid air cell, most authorities consider its anatomical development to be determined, exclusively, by the local mucous membrane.

The connective tissue and the bone tissue have likewise, each in turn, been described as the sole determining factor, and so have postnatal air pressure differences in the middle ear.

These conclusions concerning so-called normal growth, i.e., anatomical development, are based throughout on findings in histological sections from fetuses, stillborn and deceased persons of various ages.

2. *The Air Cell System.* As regards the mastoid air cell system and the causes of its final size variation several investigators have called attention to the obvious influence of heredity.

Having at hand numerical measurements of the individual air cell systems, Dahlberg and I in 1945 were able to show, in our large material of families and twins, that hereditary factors, computed singly, represent more than 84 per cent of the total causative factors. Environmental factors, likewise computed singly, were considerably less influencing, below 54 per cent. The sum of these percentages exceeds 100, which illustrates the well-known fact that factors influencing anatomical growth are working in both directions, i.e., they may stimulate and depress growth as well, thereby sometimes cancelling one another out.

It was further indicated that the final size variation was decided during prenatal life. The differing variation in uniovular and binovular twins as compared with the population seems to support this view.

3. *The Developmental Timing.* In anatomical development the timing, i.e., onset and termination of growth, must be expected to show variations, just as does the final size.

So for instance mastoid air cells are maintained to start their development approximately at birth. They are actually found by Mouret to exist in some cases as early as in the seventh fetal month.

According to my investigation in 1940 the mastoid air cell system terminates its growth at the age of about 10 in females and 15 in males. This female leadership seems to parallel other sex differences during the period of puberty, as for instance the appearance of secondary sex characteristics.

A tendency can also be shown within the rising generation in Sweden towards an increase in final size of the mastoid air cell system. This may be presumed to parallel the increase in stature lately registered in Swedish conscript soldiers.

4. *The Size Variation in the Population.* Concerning size of the mastoid air cell system measured on the x-ray film in lateral projec-

tion, the following observations can be quoted from the group of adults representative of the population:

a. The final size varied in a continuous sequence from 0 to about 25 to 30 square centimeters.

b. The mean was about 12 to 13 square centimeters.

c. The distribution of the individual sizes corresponded fairly accurately with a normal curve.

d. The individual asymmetry varied in a continuous sequence between 0 and 8 square centimeters with a mean of about 2 to 3 square centimeters.

The individual variation thus falls short of the interindividual variation, pointing to a size correlation between the two mastoid air cell systems in the same individual, i.e., they tend to approximate each other in size.

The computation of this correlation gave a confirmatory high figure of 0.86. As is well known correlation figures vary between 0 and 1.

THE SMALL MASTOID AIR CELL SYSTEM

1. *Etiologic Alternatives.* Considering that an anatomical size variation always must exist to some extent, there are only two fundamental alternatives by which to explain the total size variation of the mastoid air cell system in the population: a) On the one hand, all sizes may be caused by anatomical factors, and b) on the other hand, certain sizes may be caused by pathological factors.

From this etiological point of view the small mastoid air cell system no doubt represents the most controversial size.

Hypothetically, pathological small air cell systems may also come about in two ways: a) by a halting of growth before or during development, and b) by a diminution of the already developed large air cell system.

In turn, a diminution of a large air cell system into a smaller one may also come about in two ways: a) by a destructive acute

mastoiditis causing a disappearance of the air cell system or part of it, and b) by a productive chronic mastoiditis conceivably shrinking the periphery of the air cell system.

The observation of a gradual shrinking of the periphery of the mastoid air cell system has however not been reported, not even in manifest otitis of long duration.

Lilja and I, in 1948, published studies of a series of otitis cases radiologically and clinically followed during periods of years. In spite of the fact that chronic mastoiditis could be seen gradually developing in these cases, some of which were microscopically confirmed to have formed rough linings and sclerosing walls of the single air cells, the size of the mastoid air cell system could not be shown to shrink.

The significance of lacking reports admits naturally of arguments. Nevertheless, otitic processes, unobserved or observed, causing a diminution of the air cell system by destruction or shrinking cannot reasonably account for the small air cell systems actually found in my investigations, except possibly in rare cases.

We may disregard the fact that all sizes, from the very smallest to the very largest, occur in a continuous sequence and that, consequently, all air cell systems are smaller than the largest one.

We cannot disregard, however, that close to 50 per cent of all individuals in the population have air cell systems smaller than the average size and nevertheless show no signs whatsoever of ear pathology, neither of a destructive nor productive nature. Otoscopically, functionally and radiologically they are just as healthy as the air cell systems of the remaining 50 per cent, being larger than the average size.

We are left, then, with two final alternatives by which to explain the small air cell system:

a. The anatomical size variation.

b. The halting of growth by pathological processes before or during development.

The arguments in favor of these respective etiologies must therefore be considered.

2. *The Pathological View.* All over the otological world Wittmaack is maintained to have shown that the small mastoid air cell system represents a pathological growth.

It may therefore amaze most otologists to hear that Wittmaack himself emphatically denies that he has drawn any conclusions whatsoever concerning size of the air cell system. He stresses that his conclusions are valid for irregularity only, and that this pathological irregularity may be found in all sizes, from the very smallest to the very largest.

Wittmaack himself consequently is an anti-Wittmaackian. Even the league of Neo-Wittmaackians, if there is any, must include him out.

It would seem superfluous, therefore, to point out in this connection that Wittmaack's investigations were carried out on non-representative and not systematically collected material. In consequence, however, even Wittmaack's conclusions concerning regular and irregular growth of the air cell system must be scientifically unsubstantiated.

As is well known Wittmaack argues that the mucous membrane alone determines the development of the mastoid air cells. He maintains that the normal mucous membrane causes a regular and the pathological mucous membrane an irregular structural growth. The pathological mucous membrane is said to occur in two different patterns, both being permanent effects of infantile otitis.

Actually, no investigator has been able to show scientifically that the mucous membrane really is of exclusive importance in anatomical or pathological growth. Furthermore it has not been shown that infantile otitis has existed during air cell development in surviving individuals or that, where it does occur, it influences developmental growth at all. Finally it has not been shown that a pathological influence on developmental growth necessarily means a stunting of growth. In acromegaly, for instance, the pathological environmental factors are shown to stimulate growth.

To date, therefore, the commonly held view that small mastoid air cell systems occur by means of a pathological stunting of growth, is not only based on a misinterpretation of Wittmaack's conclusions, but also seems to lack every scientific support.

The conception that the small mastoid air cell system is pathological in any case raises the question: how small is small? What size represents the border between the anatomical and pathological air cell system? Authorities claiming a pathological nature of the small air cell system have not even been able to answer this fundamental question. The situation clearly illustrates the necessity of knowing how things are before trying to find out why they are.

3. *The Anatomical View.* As already mentioned, environmental factors are always present in developmental growth. Of course, their presence alone, even computed in percentages as done by Dahlberg and me, does not indicate whether they are anatomical or pathological in nature.

Several of my findings, however, do indicate the anatomical nature of all sizes of mastoid air cell system:

a. All sizes actually shown to exist form a continuous sequence, which is a constant characteristic of anatomical size variation.

b. The frequency distribution of all sizes in the population corresponds to the normal curves, which likewise is a constant characteristic of anatomical size variation.

c. These two characteristics were found in two separate investigations and in two separate materials, in 1940 and again in 1945, which seems to exclude coincidental findings.

d. All degrees of individual asymmetry shown to exist form a continuous sequence in a normal curve distribution, which is one more characteristic of anatomical size variation.

e. The individual asymmetry falls short of the interindividual variation, which indicates an anatomically based tendency of both air cell systems in the same individual to approximate each other in size.

f. This tendency was confirmed by the correlation figure computed, which indicates that the asymmetry does not, per se, constitute evidence of a pathological nature of the small air cell system as is usually maintained.

g. In the normal material the majority of the ears, symmetrically or asymmetrically having small air cell systems or even no cells at all,

exhibit no history of preceding otitis, nor any such signs whatsoever, otoscopically, functionally or radiologically, which indicates the absence of mastoiditis processes causing a destruction or shrinking of the air cell system.

h. The hereditary factors have a preponderant influence on final size variation, which indicates the absence of pathological factors causing a halting of growth before or during development.

Additional support may be drawn from experiences made by all of us. We all see manifest infantile otitis not resulting in a small mastoid air cell system in the adult. We even see large air cell systems develop after mastoidectomies performed in early childhood.

My investigations and the common experiences therefore strongly support the anatomical view claiming that all sizes of air cell system are anatomical in nature.

THE TYMPANIC MEMBRANE PERFORATION

1. *Etiologic Differences Between Central and Marginal Perforation.* We all admit prognostic and therapeutic differences between central and marginal tympanic membrane perforations. There seems to be etiologic differences, too, though less commonly considered.

For example, the gradual development of a central perforation is a common observation in the course of otitis media. On the other hand, no investigator has reported observing the actual appearance of marginal perforations. They have not been observed developing in the course of acute or chronic otitis media, nor to result from enlargement of a central perforation, nor from acute destructive or chronic productive mastoiditis. It is thus considered to be impossible to forecast a marginal perforation. When established it is nevertheless assumed to be the result of an otitis media.

2. *Cholesteatoma and Marginal Perforation.* For reasons which will certainly be illustrated in the next lecture, given by Prof. Ruedi, McKenzie in 1931 claimed that the cholesteatoma precedes and actually causes the marginal perforation by its expanding growth.

McKenzie also claimed that cholesteatoma has no otitic etiology but is always a genuine tumor.

I agree with McKenzie concerning cholesteatoma preceding and causing the marginal perforation. I furthermore suggest that this may be the real state of affairs irrespective of the origin of cholesteatoma, i.e., irrespective of its being a genuine tumor or not.

RELATIONSHIP OF RELEVANT SIZE AND PERFORATION

Concerning relationship between size of air cell system and development of tympanic membrane perforation, the following observations can be quoted from my investigations:

1. The size of the mastoid air cell system is of paramount significance in the pathogenesis of the tympanic membrane perforation.

2. No perforation is encountered in ears showing an air cell system exceeding the mean size of about 15 square centimeters.

3. Air cell systems falling short of the mean size do not necessarily show a tympanic membrane perforation.

4. Central perforations are encountered only in ears showing an air cell system smaller than 15 square centimeters.

5. Marginal perforations are encountered only in ears showing an air cell system smaller than 10 square centimeters.

6. The size of the air cell system in the right and left ears of the same individual may not be equal, a phenomenon referred to as asymmetry.

7. Where asymmetry is encountered it need not be associated with a tympanic membrane perforation. However, when a perforation co-exists it is found on the side with the smaller air cell system.

8. Where a unilateral perforation is visualized otoscopically it is associated with asymmetry, and the smaller air cell system is found on the side of the perforation.

RISK FIGURES

Risk figures in the population for the contracting of tympanic membrane perforations may be looked upon from two different angles.

The absolute risk run by any individual cannot be disclosed by my investigations, apart from the fact that no person seems able to contract a perforation whose air cell system exceeds 15 square centimeters.

The relative risk, however, has been computed. This risk compares the incidence of tympanic membrane perforations at different sizes of air cell systems and shows in percentages that the risk increases parallel to the decrease of the system size.

In consequence, the smaller the air cell system the lower the incidence of a spontaneous closure by cicatrization.

SIGNIFICANCE OF SIZE MEASUREMENTS

I should like to point out, finally, that it is immaterial whether the size of the measured area on the x-ray film represents the volume of the mastoid air cell system or not. Correlation figures computed between the sizes in the lateral and frontal x-ray projections were fairly high, however.

The data given, of course, do not imply that the size of the mastoid area measured, or the volume of the mastoid air cell system, is a causative factor itself in the pathogenesis of the tympanic membrane perforation.

The area measured only serves as a fairly reliable indicator of the pathogenetic possibilities, and seems to be of prognostic and diagnostic value. It is also of worth in assessing the therapeutic effect of various modern methods of operation.

CENTRALLASARETTET

XXVIII

NEUROTOXICITY OF STREPTOMYCIN AND DIHYDROSTREPTOMYCIN

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The drugs streptomycin and dihydrostreptomycin have been in large production and clinical use, the former since approximately 1946, the latter since 1950, predominantly for treatment of various forms of tuberculosis, and in combination with penicillin for synergistic effect, against other infections.

Since its early investigation, it has been known that streptomycin has an unusual selective toxicity for the VIII cranial nerve, more for the vestibular and much less for the auditory branch. The reverse is true of dihydrostreptomycin, its toxicity being predominantly on the auditory branch.[1-3]

The neurotoxicity appears to increase with each increment of the drug given, and to be irreversible, especially as far as dihydrostreptomycin and its effect on hearing are concerned. The damage to the auditory nerve sometimes appears after the drug has been stopped.

Inasmuch as the drugs are effective and needed, it is not intended to disparage them, but to call attention once more to their toxic effects, and to urge the use of plain streptomycin rather than dihydrostreptomycin, alone or in combination. The only exceptions would seem to be an allergy to streptomycin and not to dihydrostreptomycin, or bacterial resistance to the streptomycin and not to dihydrostreptomycin. Neither of these are common.

At the present time streptomycin is available, of course, from many sources. However, most combinations contain either dihydrostreptomycin and penicillin or mixtures of streptomycin and dihydrostreptomycin and penicillin. We see no reason to encourage the use of dihydrostreptomycin either alone or in combination except in the exceptional cases noted above.

Vestibular damage, though permanent in some cases, may be compensated for by increased dependence upon peripheral proprioceptive impulses and the visual mechanisms. However, perceptive hearing loss can only be aided by lip reading. Hearing impairment is more serious than balance impairment both as to work and recreation.

It is realized that those who work primarily in fields such as tuberculosis in which extended use of high doses of these drugs is common are fully cognizant of the dangers mentioned. However, it is our experience that doctors who use these drugs less frequently are less aware of them. Repeated courses of dihydrostreptomycin, though brief, may finally result in permanent VIII nerve damage.

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REFERENCES

1. O'Connor, J. B., Christie, F. J., and Howlett, K. S.: Neurotoxicity of Dihydrostreptomycin: Effects of Longer Term Therapy. Am. Rev. Tuberc. 63:312, 1951.

2. Naismith, J. T.: Deafness with Use of Dihydrostreptomycin in Tuberculosis Meningitis. Brit. M. J. 1:796, 1952.

3. Heck, W. D., Lynch, W. J., and Graves, J. L.: Controlled Comparison of VIII Nerve Toxicity of Streptomycin and Dihydrostreptomycin. Annals of Otology, Rhinology and Laryngology 62:101, 1953.

XXIX

PURE-TONE AUDIOMETRY IN CHILDREN LANTERN-SLIDES TEST

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AND

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A hearing test for young children is one of the difficult problems with which a otolaryngologist meets daily, and various methods which have been published here and abroad speak eloquently of it. For example, applications of the pupillary reflex, blinking due to sound stimuli, reflex of the eye-muscles, and E.E.G., and PGSR-audiometry and play-audiometry are chief methods. However, though each has its merits and its faults, there is at present no way but to guess the hearing threshold by putting the results of these methods together. But since it became known that hearing was impaired by dihydrostreptomycin, all the otologists have been put under the necessity of working out a way to measure the hearing of preschool children. The authors have made an effort to study these methods of audiometry. First PGSR-audiometery was tried, but it was found that this hearing test only provided material for confirming deaf-mutism or for judging the level of residual hearing in each sound area of high, middle, and low of children with various diseases of the ears. Therefore, besides PGSR-audiometry, one of the play-audiometry methods is used as a reference material for judging more exact hearing in our hospital.

The methods of play-audiometry which have been published up to date are the card picture test (Blommer), the train tunnel test (Ewing), Guildford's method and Barr's[3] method, but these tests do not have enough variety to prevent a child who is being tested from becoming tired of them, and the toys which Barr uses seem to be too difficult for a very young child. So the authors used a variation of Dix-Hallpike's peep show test.

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This material was presented before Kansai Otolaryngological Society in Kyoto on March 3, 1956.

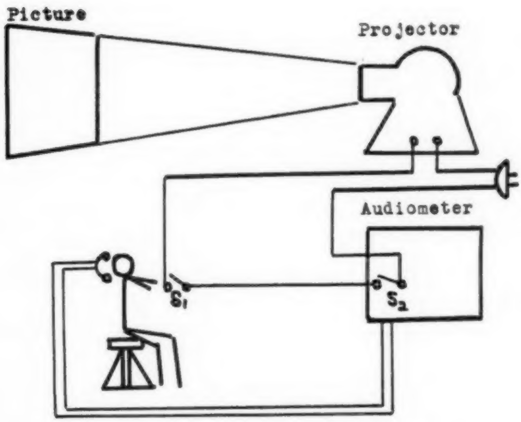
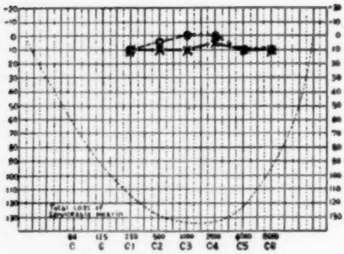


Fig. 1.—Arrangement for Lantern Slides Test.

METHOD

Various pictures are thrown on a screen in the sound-proof room by a projector when the child being tested presses the button S_1. If possible, the parent should take the child on his knee and try to interest him in looking at the lantern slides, putting the receiver to the ear to be tested. However, besides S_1 there is the switch S_2, which synchronizes with the interrupter of the audiometer, in the circuit between the source of electricity and the projector (Fig. 1), and it is so contrived that even if the child presses S_1 when the sound does not come out from the audiometer pictures will not appear, as the switch S_2 is turned off. At first, a selected pure tone of high intensity which seems to be strong enough to be audible or a tone of the highest intensity from the audiometer is delivered to the ear, and the child is asked to press S_1. When this is repeated several times, so long as normal intellect and the sound is audible, most children come to understand that a picture will appear on the screen when S_1 is pressed at the time when the sound is audible, but that it will not appear however hard they may press S_1 when the sound is not audible. By using a sort of conditioned reflex like this, the hearing threshold is decided for a given tone. Since the patients are all children, the tester is obliged to contrive some means to keep the children from becoming tired of the test. For that purpose, it is necessary to change pictures one after another. However, as it sometimes happens that the test



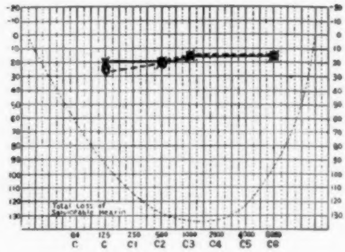
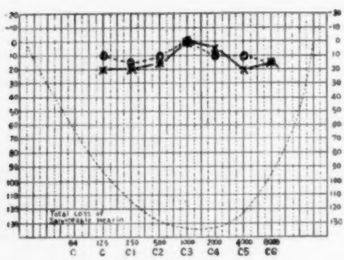


Fig. 2.—An audiogram of a six year old boy, showing a normal threshold of hearing.

Fig. 3.—An audiogram of a six year old boy whose hearing is considered normal.



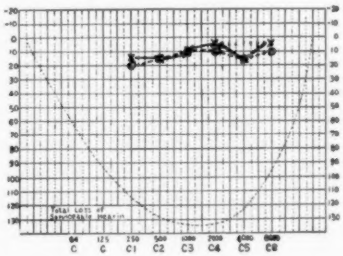
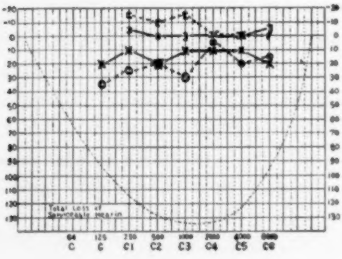


Fig. 4.—An audiogram of a five year old boy whose hearing is considered normal.

Fig. 5.—An audiogram of a five year old boy whose hearing is considered normal.



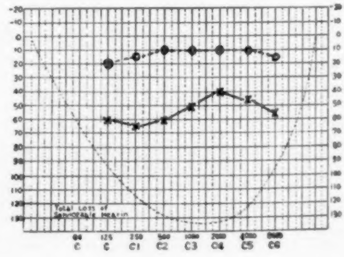


Fig. 6.—An audiogram of a six year old boy who had been complaining of hearing impairment for one week.

Fig. 7.—An audiogram of a five year old girl suffering from chronic otitis media of the left ear.

has to be discontinued because the child becomes tired of the test, it will be wise to measure the hearing threshold first for a given tone in each sound area of high, middle and low, and then to extend it to all frequency areas when there is a possibility of continuing the test.

The so-called peep-show test is disadvantageous in that the children must look at a picture in a box through a peephole; has too many needless signals, and demands the children to keep themselves in a fixed pose. Under the authors' procedure no such complicated equipment is necessary and the children can enjoy the lantern slides in an easy and free posture.

RESULTS

Since it was necessary to investigate the confidence limit of this hearing test before it was used practically, ten children with normal hearing and no family history of hearing impairment were selected for the test.

Figure 2 is an audiogram of a six year old boy, showing a normal threshold of hearing of 0 to 10 db.

Figure 3 is an audiogram at 125, 500, 1000 and 8000 cps obtained by a hearing test performed on a six year old boy, showing the threshold of 15 to 25 db on both sides.

Figure 4 is an audiogram of five year old boy. In this case, the threshold of 0 db at 1000 cps and 10 to 20 db at all other frequencies is shown.

Figure 5 is an audiogram of a five year old boy, showing the threshold of 10 to 20 db on both sides.

These four cases are audiograms of children whose hearing is considered normal, but in other normal cases too, some show the threshold of 20 to 25 db. Judging from these results, it seems that there is a difference of 10 to 15 db between the exact threshold of hearing and the threshold obtained by this method. Takeuchi[4] who had tried the peep-show test himself said that the exact threshold of hearing could be obtained by it, but it is a question whether he is right. It is important to mention here that the accuracy of a hearing test can be expected only when the testee concentrates upon hearing the sound. It is a well-known fact from our experiments and experience that a

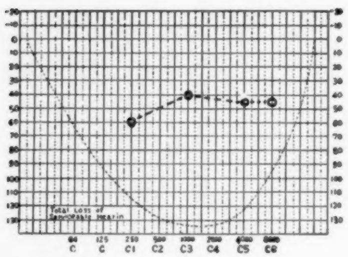


Fig. 8.—An audiogram of a three year old boy to whom dihydrostreptomycine 30 gr. had been administered.

testee does not show any response to a tone stimulus when he is absent-minded even if a tone of above-threshold is delivered to the ear. Consequently, it is questionable whether very young children can hear a hardly audible low tone near the real threshold, and it is quite natural that the threshold of hearing with play-audiometry should be higher than the real threshold by 10 db to 15 db.

Figure 6 is an audiogram of a six year old boy who had been complaining of hearing impairment for one week. The drum membrane was slightly retracted and the tonsils were enlarged and the mucous membrane of the nose showed catarrhal inflammation. In this case, even bone conduction was very smoothly measured.

Figure 7 is an audiogram of a five year old girl suffering from chronic otitis media of the left ear, showing the difference between the normal ear and the affected ear.

Figure 8 is an audiogram of a three year old boy to whom dihydro-streptomycin 30 gr has been administered. In this case only the right ear was successfully tested; the test on the opposite ear had to be given up because the boy became tired.

The cases mentioned above are some of the audiograms of lantern-slides tests which have been done. Apart from these cases, there were

some cases in which the test ended in failure as shown in Figure 8. Most of these unsuccessful cases failed because the children would not enter the sound-proof room, crying from the beginning. However, such cases were very few in children over four years of age, and there is a great possibility of success with children of three years of age if the test is performed in parts at several different times. There was no case in which the test succeeded in children under three years of age.

CONCLUSIONS

1. As an audiometric procedure for preschool children, a variation of the peep-show test was used.

2. This method is most suitable in that it does not disturb a child mentally by pain or fear and the patient does not become tired of the test owing to his interest in seeing pictures. And the equipment is very simple.

3. Since the test might have to be discontinued because the child may become tired of it if he is very young, it is suggested that at first the hearing threshold be decided rapidly for 250, 1000, 4000 and 8000 cps and then the test tones be extended to all frequency areas, if it is possible to continue the test; when the child seems to become tired of it the test should be given up immediately and reserved for another occasion.

4. It is thought that there is a difference of 10 to 15 db between the exact threshold of hearing and the threshold obtained by this method.

5. It is necessary to try once the lantern slides test when the patient is over three years of age.

6. It is necessary that the tester himself has an interest in children and is skillful in handling children.

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REFERENCES

1. Goodhill, V., Rehman, V., Brockman, S.: Objective Skin Resistance Audiometry. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 63:22-38 (Mar.) 1954.

2. Bordley, J. E., Hardy, W. G.: A Study in Objective Audiometry with the Use of Psychogalvanometric Response. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 58:751-760 (Sept.) 1949.

3. Barr, B.: Pure Tone Audiometry for Preschool Children; A Clinical Study with Particular Reference to Children with Severely Impaired Hearing. Acta Otolaryng. Supp. 121, 1955.

4. Takeuchi, K.: The Pure-Tone Audiometry in Young Children. Jap. Jour. Otol., Tokyo, 59(1):66-72 (Jan.) 1956.

XXX

TWO CASES OF NONCHROMAFFIN PARAGANGLIOMA IN THE MIDDLE EAR

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Until recently a little more than a hundred cases have been published of nonchromaffin paraganglioma in the middle ear. This lesion is better known as glomus jugulare tumor, but according to the works of Watzka,[11] Lattes[7] and Guild[6] the above mentioned nomenclature is preferable.

Usually it is difficult to make an early diagnosis of this condition and the histological picture is frequently misinterpreted. This is the reason for calling attention to this disease that is equally interesting to the otologist and to the histologist.

The tumor arises from the so-called jugular bodies described by Guild in 1941 and more thoroughly in 1953.[5,6] Guild states that histologically the glomus jugulare closely resembles the carotid body. Each glomus (or paraganglion) consists essentially of numerous small blood vessels, capillaries or precapillaries in size, and of epithelioid cells in the spaces between the blood vessels. The proportion of epithelioid cells to blood vessels may differ considerably in individuals.

Somewhat more than half of the glomus formations found are in the adventitia of the dome of the jugular bulb, along the jugular fossa part of the course of either the nerve of Jacobson (tympanic branch of the glossopharyngeal nerve) or the nerve of Arnold (auricular branch of the vagus); another fifth are in the osseous canal (tympanic canaliculus) through which the nerve of Jacobson enters the middle ear from the jugular fossa. About one-fourth of the ears have a glomus formation in the mucosa of the cochlear promontory, along the tympanic plexus portion of the course of the nerve of Jacobson.

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In none of the locations in which a glomus jugulare may be present is the structure of constant occurrence.

Jugular bodies differ in size from about 1.5 mm in the longer dimension to about 0.1 mm.

"Neither race, sex or side of head is significantly related to the number, distribution, size or histologic appearance of jugular bodies found in the material examined. Age does seem to be related to the number of jugular bodies present, with the period of mid-adult life that of the highest incidence.

"The differences in order of clinical symptoms that have been reported for patients with glomus-jugulare tumors, also the differences in apparent site of origin found at operations to remove such tumors, can all be satisfactorily explained by the differences in the observed locations of normal jugular bodies, had they undergone an abnormal enlargement."

The glomus jugulare tumors are generally slow-growing. Metastasis from such growth is rare. The initial symptom may appear at any age. Although the symptomatology varies according to the location of the tumor, the middle ear is a common site of invasion with ensuing lesion of the facial nerve and the cochleovestibular apparatus. By expanding within the jugular fossa the tumor often encroaches upon the IX, X and XI cranial nerves (the syndrome of the jugular foramen). By further extension into the cranial cavity even the XII and more rarely the V and the VI cranial nerves may become paralyzed.

The symptoms are those usually met with in affections of the middle and the inner ear, namely, impaired hearing, tinnitus and dizziness. In addition headache may supervene.

At an early stage the otoscopy may reveal a reddish and bulging drum. Later on aural discharge is a common finding including polypoid masses in the ear canal showing a marked tendency to bleed after the slightest trauma. An additional weakness of the face may be found to a varying degree. The long duration, also, of the symptoms is a characteristic feature, as are a recurrence of polypoid masses in the ear canal after repeated removals, and the involvement of cranial nerves caudal to the facial.

REPORT OF CASES

Both of the following case records have been presented before the Danish Otolaryngological Society; Case 1 in 1953 by Richter Jorgensen and Case 2 in 1956 by the author.

CASE 1. L.C.H., a 68-year-old widow. Since 1944 she has complained of tinnitus and impaired hearing of the left ear. In 1947 an acute otitis media was diagnosed on the left and a paracentesis was made. Two weeks later the ear was dry but the hearing of the left ear was still bad. In March 1952 she complained of increasing tinnitus and hardness of hearing and the examination by the ear-nose-throat specialist now revealed a red and bulging left drum. A repeated paracentesis was followed by marked bleeding. There was no pain. In October and again in November 1952 polypoid masses were removed from the left ear canal. On both occasions a histological diagnosis of "polypus granulomatosus" was made. Shortly afterwards she complained of vertigo. A spontaneous nystagmus towards the unaffected ear was disclosed. The left ear was completely deaf. Two days later a left-sided facial palsy was noted and the patient was admitted to the ear, nose and throat department of the State Hospital in Sonderborg.

On examination polypoid masses in the ear canal were found. The deafness of the left ear and the nystagmus to the right remained unchanged. There was no caloric response from the left labyrinth. The paresis of the facial nerve involved the three branches being less pronounced in the frontal region. No affection of the remaining cranial nerves and no cerebellar symptoms were found. The cerebrospinal fluid was normal. Roentgen of the ears showed normal pneumatization without any destruction.

No relevant abnormalities were disclosed on ophthalmological and neurological examinations.

A left-sided radical operation was performed. In the mastoid the cells were filled with brownish fluid; no destruction was seen. In the middle ear and the attic grayish-red, dense granulation tissue protruded. The slightest touch of these was followed by intense hemorrhage. All granulation tissue was removed and hemostasis was eventually satisfactory. The postoperative course was uneventful, the cavity soon dried and became epithelialized. The paresis of the facial nerve and the nystagmus disappeared in a few days. The deafness of the left ear, however, remained unchanged.

The first histological descriptions were unsatisfactory. Therefore the removed granulation tissue was examined several times and by different histologists. (The microphotos were made by the pathological institute of the Kommunihospital, Copenhagen.)

Abstract of Description 1: Small irregular groups of tumor cells with pycnotic nuclei. The basal tissue is remarkably vascularized and reminds one of granulation tissue. It is impossible to decide between a mesenchymal or an epithelial origin. Histological diagnosis: malignant tumor.

Description 2: H.D. Tumor tissue (malignant??).

Description 3: H.D. Granulation tissue with changes suspected of carcinoma.

Description 4: H.D. Granulation tissue with irregularly regenerating epithelium.

Description 5: H.D. Granuloma teleangiectaticum, presumably a glomus jugulare tumor.

On follow-up in March 1957 she felt completely well. No paresis of the facial nerve. The left ear remained deaf. The operative cavity in the temporal bone was dry and epithelialized.

Case 2. H.C.M., a 69-year-old former laborer. Since 1945 he had complained of impaired hearing of the right ear.

In 1952 the hardness of hearing had increased considerably and he was examined at the Ear, Nose and Throat Clinic for out-patients of the State Hospital, Sonderborg.

Otoscopy revealed on the right side a bluish-red bulging tympanic membrane without any perforation. Conversational voice, but not whispered voice was heard when spoken close to the ear. On tubacatheterization of the right ear hemorrhage occurred in the ear canal. The hearing remained unchanged.

A diagnosis of angioma of the right middle ear was made.

The patient did not return until 1956.

About a year after the last examination a right-sided facial paresis had slowly developed; but as he still felt well he did not consult a specialist.

In September 1956 he had noticed some difficulty in swallowing for the first time and he experienced a sensation of heaviness in the right side of the tongue. An ear, nose and throat specialist found no vestibular response from the right ear; polypoid masses were found in the right ear canal. During removal of this, aboundant hemorrhage occurred both through the ear canal and through the eustachian tube.

Histological examination revealed tumor tissue with remarkable vascularization. In the spaces between the blood vessels big polymorphical, hyperchromatic cells were seen. A histological diagnosis of presumable glomus jugulare tumor was made.

At that stage the patient was admitted to hospital.

Beside the above mentioned tumor tissue in the right ear canal and an abolished function of the right labyrinth, a pronounced right-sided jugular foramen syndrome (paresis of the IX, X and XI cranial nerves) and also a paresis of the VI and VII right and the XII left cranial nerves were found on examination.

Roentgen of the skull showed a destruction of the bone in the region of the jugular foramen localized mainly to the petrous part of the temporal bone.

No signs of metastasis could be demonstrated in the lungs or in other organs.

The involvement of so many cranial nerves—and even one on the contralateral side—indicated an expansion of the tumor into the cranial cavity to the extent that the operative risk was hazardous.

Therefore the patient was transferred to the radiation center, Copenhagen, for radiotherapy. 2125 and 1870 r were given on each field.

COMMENT

Both case reports would seem instructive. Presumably, in Case 1 the primary location of the tumor has been in the middle ear itself, and the removal has taken place in due time to be a complete one.

In Case 2 a diagnosis was not, unfortunately, made when he was seen for the first time at the hospital. Possibly it would have been removable at this juncture. Characteristic, however, in this case are the very slow progression and the occurrence of the jugular foramen syndrome.

Both cases would seem to stress the importance of making so early a diagnosis as to make surgical intervention possible without undue risk to the patient.

In order to make a diagnosis as early as possible bioptic examination would seem advisable of any tissue in the ear canal suspected of this type of neoplasm.

Removal of large glomus jugulare tumors and especially those with neurological signs requires opening of the upper neck and of the cranial cavity, and in these cases the mortality is high.

In such cases and in those also in which surgical removal could not be completed, roentgen therapy is the treatment of choice.

The effect of this type of treatment is commonly taken to be questionable; but, according to Capps,[2] it would seem that deep radiation treatment may, after all, give satisfactory results.

For further information in this field references are made to the works of Rosenwasser,[10] Berg,[1] Zettergren and Lindström,[12] Cleary,[3] Lundgren,[8] Crue and Poulsen,[9] and co-operators.[4]

SUMMARY

Two cases of nonchromaffin paraganglioma (glomus jugulare tumor) in the middle ear are reported. In Case 1 the lesion was removed surgically. In Case 2 the tumor had expanded to the extent that the operative risk was too hazardous. The patient was treated with roentgen.

The interpretation of the histological picture, particularly of the former case, caused considerable difficulty.

SUNDQUISTGADE 4

REFERENCES

1. Berg, N. O.: Tumors Arising from the Tympanic Gland (Glomus Jugularis) and Their Differential Diagnosis. Acta Path. Microbiol. Scand. 27:194-221, 1950.

2. Capps, F. C. W.: Treatment of Glomus Jugulare or Tympanic Body Tumors of the Middle Ear and Petrous with Particular Reference to Radiotherapy. Proc. Fifth International Congress of Oto-Rhino-Laryngology, Amsterdam, 1953, pp. 273-282.

3. Cleary, J. A.: Nonchromaffin Paraganglioma (Glomus Jugulare Tumor) of the Middle Ear. Arch. Otolar. 56:378-384, 1952.

4. Crue, B. L., Freshwater, D. B., Shelden, C. H., Hunnicutt, L. G., and Johnson, F.: Syndrome of the Jugular Foramen. Arch. Otolar. 63:384-391, 1956.

5. Guild, S. R.: A Hitherto Unrecognized Structure, the Glomus Jugularis in Man. Anat. Rec. (Suppl. 2) 79:28, 1941.

6. Guild, S. R.: The Glomus Jugulare, a Nonchromaffin Paraganglion in Man. Annals of Otology, Rhinology and Laryngology 62:1045-1071, 1953.

7. Lattes, R.: Nonchromaffin Paraganglioma of Ganglion Nodosum, Carotid Body and Aortic-Arch Bodies. Cancer 3:667-694, 1950.

8. Lundgren, N.: Tympanic Body Tumors in Middle Ear: Tumors of Carotid Body Type. Acta Oto-laryng. 37:367-379, 1949.

9. Poulsen, J. P. V.: Middle Ear Tumors Proceeding from the Glomus Jugulare. Acta Otolaryng. 46:58-63, 1956.

10. Rosenwasser, H.: Carotid Body Tumor of the Middle Ear and Mastoid. Arch. Otolar. 41:64-67, 1945.

11. Watzka, M.: Die Paraganglien. Handbuch d. mikr. Anat. d. Menschen. Bd. 6. T. 4:262-308, 1943.

12. Zettergren, L., and Lindström, J.: Glomus Tympanicum. Its Occurrence in Man and Its Relation to Middle Ear Tumors of Carotid Body Type. Acta Path. Microbiol. Scand. 38:157-164, 1951.

XXXI

APPLIED PHONIATRICS

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SPEECH AFTER REMOVAL OF THE UPPER JAW; DEPENDENCE OF CONSERVATION OF FUNCTION AND APPEARANCE UPON PATHOLOGY

Resonance is as essential to the voice as to the violin.

When the upper jaw is removed for malignant disease speech at once becomes as unintelligible as that of a person born with cleft palate; in fact, even less so because there has been no daily practice from childhood in overcoming the defect. True, articulation may be readily restored by means of a prosthesis, which is usually fitted as soon as possible after the conclusion of the operation to combat cicatricial contraction. It is, however, an established principle of all conservative and restorative surgery that transference of living tissue is preferable to substitution of inert material, which has a tendency to set up irritation eventually. Moreover, primary repair yields better results than secondary plastic procedures, especially in dealing with facial mutilations.

I have therefore employed, after preliminary ligation of the external carotid above the superior thyroid and lingual branches, the following modifications.

The upper part of the cutaneous incision is carried along the grey line of the lower eyelid and deepened between the cilia and the tarsal plate. The motor nerve filaments and lymphatics of the lower eyelid are thus preserved intact. During maxillary resection it is often possible to conserve the palatal mucoperiosteum; if so, this is sutured to the homolateral septal mucosa which is detached and turned downwards to reconstruct the antral floor, as soon as the maxilla has been removed.

From the Hospitals of Halifax and Huddersfield, England.

Read at the Sixth International Congress of Otolaryngology, Washington, D. C., May 1957.

If the nasal septum is healthy, the greater part of it is reflected laterally as an osteoplastic flap, pedicled posteriorly to retain its blood supply, and sutured to a suitably placed drill-hole in the zygomatic region or to the temporal fascia. Stainless steel wire is suitable for the purpose. Sufficient septal tissue is left anteriorly to support the so-called nasal pyramid. A small residual aperture anteriorly in the palate leaves scope for dental treatment and permits of adequate inspection of the cavity, access to which is also facilitated by reflection of the septum.

In practice the treatment and measures of repair vary with the pathology of the tumor, without due consideration of which any attempts at restoration would be idle. Precocious invasion of glands, for example, is suggestive of one of the more anaplastic growths, which respond at first dramatically to irradiation but tend to recur and to cause the death of the patient within a few months.

More suitable for surgical treatment are cases of papillary carcinoma, the development of which is so often the last chapter in the life history of recurrent nasal polypi: a picture is revealed by biopsy of compact columns of epithelial cells amid connective tissue, containing pseudo-acinar spaces lined by an epithelium which is partly of stratified squamous and partly of columnar ciliated type. Adamantinomas, in which cribiform epithelial masses enclosed by palisades of basal cells, derived from dental tests, are locally malignant only. These are perhaps the best of all for restoration; their tendency to recur and kill the patient within ten years justifies maxillary resection at the earliest possible date.

It has been stated that adenocarcinomas which arise from the goblet cells of the mucous membrane of the antrum, especially in the vicinity of the intraorbital canal, never metastasize, but in a recent case radiological evidence of secondary deposits in the spine two years after histology was evidence to the contrary sufficiently convincing to the family doctor and friends, though no postmortem examination was made. In suspected cases of this type, in which the presenting symptom is neuralgia, instillation of lipiodol reveals or excludes early carcinoma.

Anomalies of dentition suggest that an osseous swelling presenting at about twelve years of age is due to fibrous dysplasia rather than to osteosarcoma. The prognosis of osteoblastoma is worse than that of osteoclastoma. In bony tumors the palatal mucoperiosteum is seldom

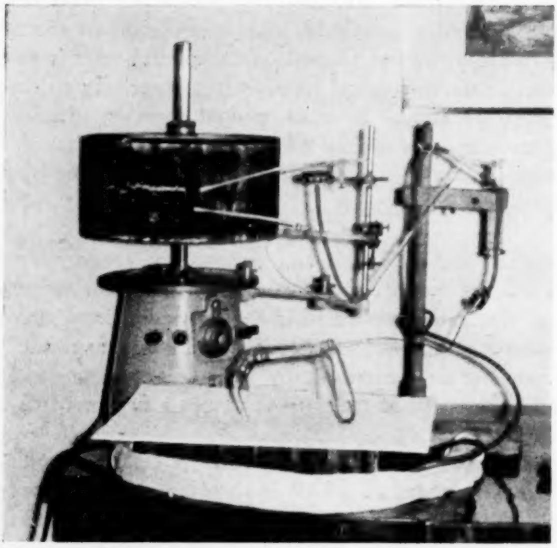


Fig. 1.—Apparatus for recording the movements of the wall of the chest. A Model 3 Laryngograph is also shown.

available for repair but the turbinals, bent outwards, may afford valuable support for the orbit. Because the patients are young, all possible measures are indicated to mitigate deformity. In two cases operated upon by me over twenty years ago, mutilation is negligible; one of the patients is now a grandfather. In leontiasis ossea the tendency seems to be towards extension to the other side. Sometimes one inherits cases resistant to treatment of which current histology remains problematical; renewed biopsy is not always desired: the large tumors and severe deformities encourage one to be drastic at the outset of any new case. It is very necessary to exclude adrenomedulloblastoma and multiple myelomatosis.

Two malignant melanomas which had not responded to irradiation were successfully excised, but in each of these the septum was invaded by the growth so that it could not be utilized in repair; in one of them deformity was severe.

In the celebrated case of maxillary resection performed by Drs. Bryant and Keen on President Cleveland on board the yacht "Oneida"

(to avoid press publicity), the floor of the orbit was left intact and there was no recurrence after fifteen years; the tumor was regarded as a sarcoma.

Commentaries on leading cases by Syme[9] (1829) and Lizars[7] (1830) have the authentic ring of true surgery. Removal by Fergusson[2] (1842) of a supposed maxillary osteosarcoma of eight years' duration from a girl of twelve, who bore the operation with great fortitude, lasted sixteen minutes. In a monograph of 112 pages, Heyfelder[4] (1857) reviewed all the literature up to that date and advocated the chain saw, with other refinements of technique.

Many suggestions have been put forward, since that date, for improvement of maxillary resection and for mitigation of deformity, but the operation fell into desuetude at the advent of radiotherapy. Renewed interest is reflected in an article by Schoolman (1956), who observes that a factor which adds to the confusion arising from the diverse methods of selecting cases is the lack of agreement on classification of tumors; for example, lympho-epithelioma and undifferentiated cell carcinoma of the nasopharynx were considered epithelial tumors in the United States but were grouped with the reticulum celled carcinomas by the Scandinavians. He adds that in the last ten years at the University of Illinois the rate of cure in maxillary antral tumors has risen from the generally reported rate of 32 to about 50 per cent.

In certain advanced cases the orbital contents must be exenterated, both halves of the maxilla removed or other grave sacrifices made. Generally, thanks to the pioneer work of generations of surgeons, results comparable with those of treatment of new growths in other parts of the body can be obtained. With these successes, preservation of function, particularly of speech, is not an incompatible aim.

An alternative method is to line the cavity with Thiersch grafts. This limits contraction of adherent and indolent scars but affords little support for the orbital contents. Just as the former practice of leaving infected mastoidectomy cavities open was discontinued, in most clinics, before the introduction of antibiotics, so with improvements in the technique of irradiation, the need for leaving the cavity formed by maxillary resection open may be less strongly felt. It is most desirable that the first operation should be the last.

Whenever malignant disease is considered it is satisfactory to recall that hypernophroma is sometimes followed by one metastasis and no more.

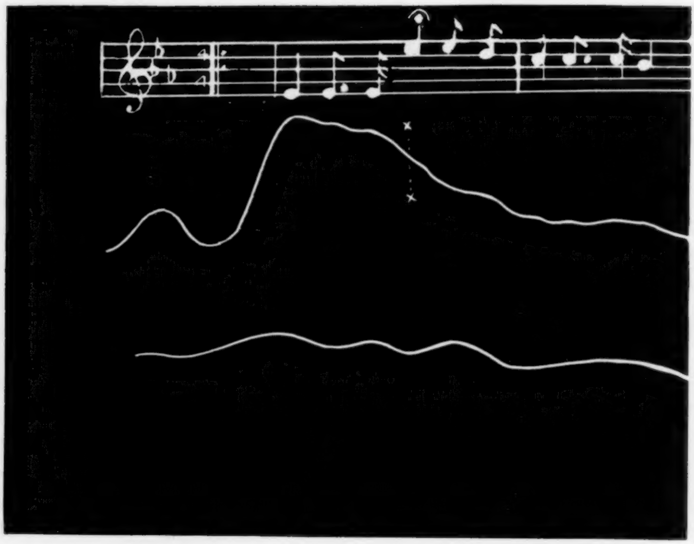


Fig. 2.—Stethy- and laryngo-graph tracings of an octave leap by a baritone voice, showing almost constant rate of deflation of chest. The model 3 laryngograph is as yet capable of only the crudest recordings.

OBSERVATIONS ON THE RIMA GLOTTIDIS

Whether the rima glottidis assumes the form of an isosceles triangle or that of a rhomb an admirable adaptation to the needs of respiration may be discerned. In fact, the following trigonometrical law may be enunciated: the cross sectional area of the rima varies with the sine of the angle of separation of the vocal folds. It follows that the area is greatest when they diverge to a right angle. Another geometrical theorem proves that the area of the aperture increases in proportion to the square of the length of the vocal fold; this helps to explain the effects of age and sex upon the range and timbre of the voice.

The glottis contracts synergically during expiration, as if to retard the elastic recoil of the lungs and curtail the postexpiratory pause. This involuntary monitoring of the respiratory wave is probably conducive to a rhythm economical of effort and varies according to the oxygen requirements of the body. It may be seen to continue

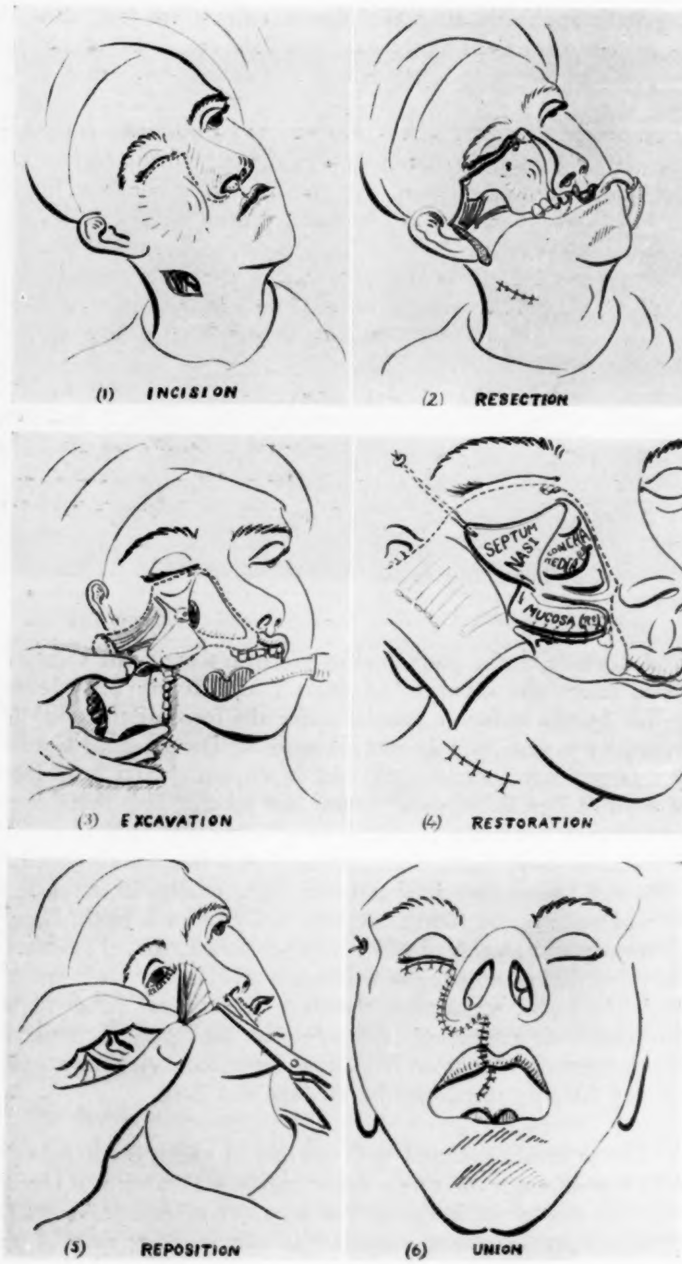
under light anesthesia after the planter reflexes are lost, until the knee jerks and reactions of the pupils to light are abolished. Would a purposeless synergy be so constant?

Normally, the expiratory phase of the modulated respiratory carrier wave is employed in speech. In sighing, and in certain cases of bilateral recurrent nerve paresis, phonation occurs during inspiration. In a recent case of left recurrent nerve paralysis I observed that it was the paretic vocal fold that vibrated and gave rise to moaning sounds as the other fold approached it, during recovery from a general anesthetic. In a third case of this kind, during conscious phonation, the active side vibrated more than the paretic one; quiet respirations were 12 to the minute.

Speech, being an ultimate acquisition after millions of eons of evolution, is produced by structures which belong primarily to the respiratory and alimentary systems. Reflex closure of the glottis during deglutition is essential to life. Speech is closely linked to hearing, depends upon memory, is swayed by the emotions and is subject to perpetual interplay of internal secretions of endocrine glands.

According to the traditional myo-elastic theory, the vocal folds vibrate under the influence of expired air, having been brought together by the adductor muscles under the force of the will. The revolutionary view so ably put forward by Dr. Edouard Garde[3] is based largely upon stroboscopic and experimental data provided by the work of Drs. R. Husson,[5] Laget[6] and others. It is that impulses pass along the recurrent nerves at audiofrequency, at least so far as the so-called chest register is concerned, in a manner corresponding to one well known theory of audition. The falsetto in the male and the head voice in the female are admittedly of such pitch, being of a frequency of upwards of 450 cycles per second, that the refractory interval of the neuromuscular junction would prevent their transmission. The higher tones must therefore be produced by di-, tri- or even tetraphasic vibrations. According to this theory the range of a singer's voice depends upon individual chronaxie. Arguments to the contrary are ably marshalled by Dr. van den Berg.

I have satisfied myself with the aid of stethygraph recordings that a trained singer can sing scales or leap an octave without changing either the rate of expiration or the intensity of sound: increases in sound pressure are due to variations in accent rather than of pitch,


(1) INCISION
(2) RESECTION
SEPTUM NASI
CONCHA MEDIA
MUCOSA
(3) EXCAVATION
(4) RESTORATION
(5) REPOSITION
(6) UNION

as in the music of the bag-pipes. To postulate a constant intratracheal pressure clarifies theory of emission of the singing voice as much as practice in *legato* passages improves the performance of the singer.

The histology of the vocal folds suggests significant elastic recoil. The action may resemble that of a ball beaten every alternate bounce or swing intermittently propelled. The two main theories of phonation would thus to some extent be reconciled.

Whatever theory of sound emission is held, eventual transformation of tissue oscillations into sound waves, transmitted by air to the endolymph of the listener, must occur or interest would languish.

Persons from whom the larynx has been excised have to learn a new method of speech. A patient whose larynx I removed four years ago is particularly competent in long-distance telephone calls. The possibility that filaments from the recurrent nerve are concerned in pharyngo-esophageal speech is worth bearing in mind during laryngectomy.

Quaternary alkaloids and synthetic relaxants act on the respiratory mechanism first upon the larynx, as shown by cessation of the synergic respiratory movement described above. Secondly, they act upon the intercostal muscles, as shown by tracheal tugging. Thirdly, they paralyze the diaphragm until with a little help from the anesthetist the effects wear off and the normal mechanism comes back to play.

How may these observations be applied? In diagnostic and operative peroral endoscopy relaxation may be prolonged at a threshold level which affords tranquil access to the parts while the action of the diaphragm is only slightly depressed, preferably by the setting up of an intravenous drip. My most expert anesthetists find that 500 milligrams of succinyl chloride (A & H, Scoline brand) in a litre of normal saline will give complete relaxation for periods sufficient for the operations to be carried out without haste, in a series of cases. It is thus possible to avoid the frustration of receiving histologic reports repeatedly negative, in conflict with the clinical diagnosis,

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Opposite Page—A series of line drawings illustrating reconstruction of the maxillary antrum after removal of the upper jaw for malignant disease. The ideal incision traverses the grey line of the lower eyelid, reflecting the eyelashes, orbicularis muscle fibres and lymphatics with the flap: inferiorly it skirts the philtrum of the upper lip.

due to submission of inadequate specimens for biopsy. Laryngectomy or other treatment may then be carried out without delay, as indicated.

To the clinician, observation of Nature is all-sufficient, but to convince all concerned pharmacologic proof is necessary. Scientific progress often depends on new apparatus. I have accordingly constructed prototype models of an intralaryngeal recorder of laryngograph. A kymograph was kindly lent by Professor W. A. Bain of the University of Leeds and his assistants, Drs. G. A. Mogey and K. A. Exley, who gave most helpful advice. On December 22, 1956, I succeeded in making a crude tracing of my own vocal folds in action. Work is in progress. Difficulties are merely technical.

Stimulating as is the deadline imposed by the four-yearly International Congress of Otolaryngology, for which this contribution has been prepared, there is enough scope for investigation in applied phoniatrics to occupy a lifetime.

SUMMARY

To preserve speech after resection of the maxilla the greater part of the nasal septum may usefully be reflected laterally as an osteoplastic flap. This modification and an incision carried along the grey line of the eyelid minimize mutilation and so render the operation more applicable to incipient antral tumors, especially of those types in which metastasis is rare.

The area of the rima glottidis varies perpetually with the sine of the angle of divergence of the vocal folds. A more static ratio, depending partly on age and sex, is that the area of the glottic aperture varies with the square of the length of the vocal fold. Synergic expiratory convergence monitors the respiratory rhythm, or is in harmony with it.

Under general anesthesia the selective action of quaternary alkaloids and synthetic relaxants was applied to intralaryngeal operations. These were performed at a level at which the myoneural junctions of the diaphragm were active or only partially blockaded.

An intralaryngeal recorder enabled a crude tracing of the contractions of one's own glottis to be made on a kymograph. Further experiments are in progress.

GREEN LEAS, SAVILE PARK, HALIFAX, YORKSHIRE

REFERENCES

1. Berg, Jw. van den: On the Myo-elastic and Neurochronaxic Theories of Phonation. Revue de Laryngol., Otol., Rhinol. 75:494-512, 1954.

2. Fergusson, W.: Osseous Tumour of the Upper Jaw—Excision of the Superior Maxillary Bone. Provincial Med.-Surg. Jour. 3:440-441, 1841-2.

3. Garde, E.: The Voice. Presses Universitaires de France. In Que Sais-Je series. 108, Boul. Saint Germain, Paris. Price 3/6d. 1954.

4. Heyfelder, O.: Resection of the Upper Jaw. A Monograph. Georg Reimer, Berlin, 1857.

5. Husson, R.: Study of the Fundamental Physiological and Acoustical Phenomena of the Singing Voice. Paris. Thesis: Faculty of Sciences of Paris, edited by La Revue Scientifique, 4, rue de Pomereu, Paris (XVIe), 1950.

6. Laget, P.: Experimental Reproduction of the Vibration of the Vocal Cords in the Absence of All Current of Air, by Electrical Stimulation of the Recurrent of the Dog, with Stroboscopic Observation of the Laryngeal Response. Revue de Laryngol., Otol. et Rhinol. 74:133-142, 1953.

7. Lizars, J.: Excision of the Upper Jaw Bone. Lancet 2:54-55, 1830.

8. Power, D'A.: Removal of the Upper Jaw: an Historical Operation. Brit. Jour. Surg. 2:780-785, 1937.

9. Syme, J.: Superior Maxillary Bone Excised. Edin. Med. Surg. Jour., R.C.S. Library, London, 32:218-220, 1829.

XXXII

THE NEUROLOGY AND FUNCTION OF THE PHARYNX, AND ITS POWERS OF COMPENSATION IN PARALYSIS

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Common complaints, like nasopharyngeal catarrh, unexplained difficulties with the act of swallowing, or in clearing the voice, slight speech defects, snoring, mouth-breathing, and problems of conductive deafness, may sometimes be due to unsuspected partial paralysis of the pharynx. Cases will be discussed where undiagnosed paralysis has been present for many years; in one case, for 30 years. These cases illustrate the remarkable powers of compensation shown by paired muscle groups in the pharynx, when one side has been paralyzed for a long time.

When measles or chicken pox affect a family, sometimes one or other child may escape the infection, although close contact may have been maintained throughout the whole course of the disease. Is it not possible that such children may have contracted the disease, at the time of the family infection, in so attenuated a form as to pass unnoticed? Do not children, in a similar way, inoculate themselves at school? Are not some children inoculated in this way with other virus diseases like poliomyelitis? Is it not possible that milder forms of undetected virus infection may leave behind some slight weakness of muscle and function, accounting for some of the symptoms described above?

SUBCLINICAL PARALYSIS

Sharrard[8] examined the brain and spinal cord of a woman aged 23 years, who died 22 months after the onset of poliomyelitis. He found that in some muscle groups where recovery had apparently been complete (Medical Research Council grading, power 5, i.e., nor-

From the London Hospital. Read at the Sixth International Congress of Otolaryngology, Washington, D. C., May 1957.

mal strength of muscle), 50 per cent of the large motor cells normally supplying these muscles had been completely destroyed.

This very important evidence strongly suggests that some muscles damaged by poliomyelitis, in the soft palate, pharynx or larynx, may apparently recover, leaving no detectable clinical paralysis, and yet may remain permanently weakened to some slight extent. Equally, during the acute phase of poliomyelitis, there may never have been any clinical evidence whatever of paralysis in the pharynx, even though this may have been carefully looked for at the time, and yet some palatal muscles may have had up to 50 per cent of their large motor cells destroyed. The efficiency of these important muscles must have been impaired to some extent, even if paralysis is not evident clinically. Could not this condition of subclinical paralysis account for cases of persisting obstinate postnasal catarrh, difficulities with palatal speech, snoring, mouth-breathing at night, and some forms of catarrhal deafness, for which there is no obvious explanation? (Fig. 1)

PSEUDOPARALYSIS

Scarring of the soft palate following tonsillectomy or gummatous ulceration may limit movement of the palate, causing symptoms due to impaired function. Such scarring can cause symptoms very similar to paralysis.

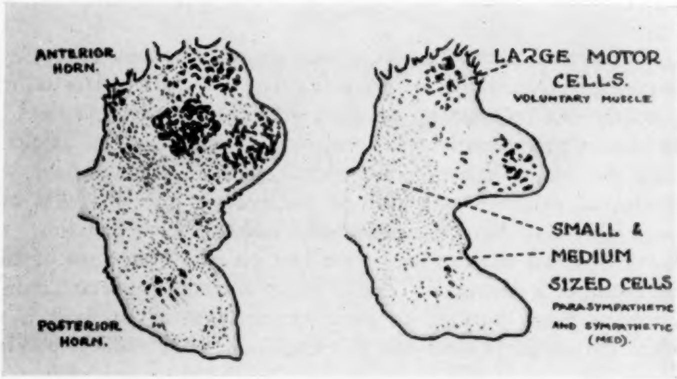
Partial cleft palate prevents proper closure of the nasopharyngeal sphincter and so produces symptoms very similar to those found in paralysis.

Hyperplasia of tonsils buried in the palate, and hyperplasia of adenoid tissue can limit movements of the soft palate and give rise to symptoms similar to those caused by paralysis.

NEUROLOGY

The cranial part of the spinal accessory nerve (XI cranial nerve) is the main motor supply to the pharynx. The peripheral pathways are different, but the central connections are probably identical for most of this muscle (Fig. 2).

In the medulla, the nucleus ambiguus is the common motor nucleus for the IX, X and XI cranial nerves. The fibres of the cranial



NORMAL POLIOMYELITIS

Fig. 1.—Correlation of clinical paralysis with motor cells. Woman, 23 years of age, died 22 months after onset polio. Fifth lumbar spinal segment. After Sharrard.

part of the spinal accessory (XI nerve) run mainly in the vagus (X nerve). The main peripheral motor nerves of the vagus carrying XI nerve fibres are

1. *the pharyngeal nerve* supplying all the muscles of the soft palate (with the exception of the tensor palati which is supplied by the mandibular division of the V cranial nerve via the otic ganglion), and all three constrictors of the pharynx, including filaments to the cricopharyngeus muscle.

2. *the superior laryngeal nerve,* supplying, through its external branch, the cricothyroid muscle, and sending filaments to the crico-pharyngeus; also possibly through motor fibres in the internal (mainly sensory) branch to the transversus arytenoideus.

3. *the recurrent laryngeal nerve* supplying not only the main muscles of the larynx, but also the cricopharyngeus muscle.

FUNCTION: SWALLOWING, RESPIRATION, SPEECH

The pharynx is essentially a simple muscular tube which opens above into the nasopharynx, and below into the esophagus. There are two further important openings in the anterior wall of this tube,

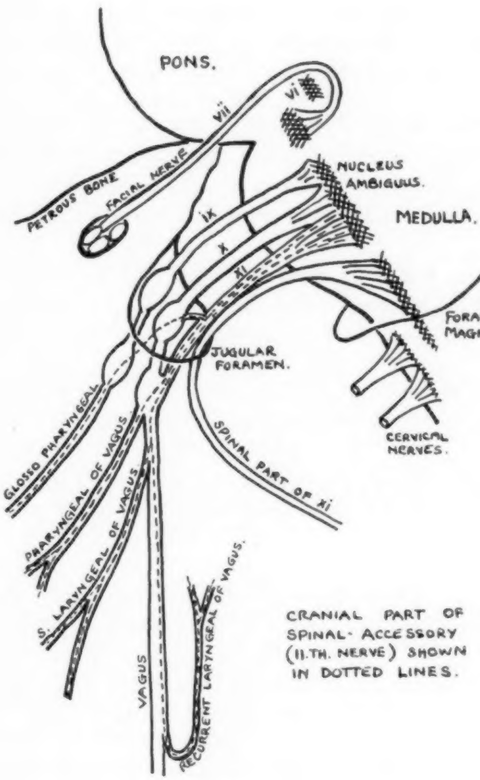


Figure 2

the mouth, and the larynx. Each of these four openings, one above, one below, and two anteriorly, is really a muscular sphincter, enabling the openings to be shut off, or opened, as required in the efficient operating of the essentially important functions of swallowing, respiration and speech. All these sphincters have a rather complex arrangement of their muscles.

The second stage of swallowing, which is involuntary, when once initiated, takes place in the pharynx. This stage begins with the closure of the faucial (mouth) sphincter, and ends with the entry of the food or fluid into the esophagus (relaxation of lower sphincter of pharynx).

The Beginning of Swallowing. There is a pause in mastication. The anterior part of the tongue is raised and pressed against the hard palate, the movement commencing at the tip and spreading rapidly backwards. To squirt fluids, the tongue forms a chute or gutter. The hyoid bone moves forwards and upwards (mylohyoids, geniohyoids, digastrics, stylohyoids). The forward movement is important. The co-ordinated movement of the tongue, and the two palatoglossus muscles, closes the faucial (mouth) sphincter. The tongue is drawn upwards and backwards into the pharynx (styloglossus).

The anterior pillars of the fauces contract (palatoglossus muscles) and meet the tongue behind the food, which is thus passed into the pharynx.

The act of swallowing is continuous. Initiation of the first stage inevitably leads to the completion of all three stages.

The nasopharyngeal sphincter is closed by the raising of the soft palate (levators) and the approximation of the paired palatopharyngeus muscles.

The larynx is drawn upwards and forwards under the tongue, and this action, combined with the movement backwards of the tongue to impel the food into the pharyngeal tube, causes the epiglottis to lie pressed horizontally under the tongue. The larynx being, as it were, safely tucked up under the tongue out of the way.

The laryngeal sphincter closes and pulls the arytenoid cartilages under the horizontal epiglottis until they lie in close contact with it, thus securely preventing entry of food, liquid or solid, into the larynx.

The forward movement of the hyoid opens the pharynx for the passage of food, and the superior, middle, and inferior constrictors pass the food on; a wave of contraction being preceded by relaxation.

The lower sphincter of the pharynx (cricopharyngeus) relaxes, and opens. The food passes on into the esophagus. This cricopharyngeus sphincter is not the upper sphincter of the esophagus, but is the lower sphincter of the pharynx.

During respiration, between the acts of swallowing, the larynx descends and opens, the tongue moves forwards to its relaxed position,

and the epiglottis returns to its upright position. The lower pharyngeal sphincter closes to shut off the esophagus. The nasopharyngeal sphincter relaxes to allow nasal breathing. The tensor palati holds the soft palate to prevent flapping.

During speech, the various sphincters stay open or closed in much the same position as they do for respiration, but their lumen alters, and some of their muscles contract or relax to supply the necessary quality and pitch for speech. The tongue, soft palate muscles, pharyngeal muscles, and laryngeal muscles all play their integral parts in the production of speech.

THE NASOPHARYNGEAL SPHINCTER

The muscles concerned are the paired levators, the paired tensors, the paired palatopharyngeus muscles, and, to a lesser extent, the upper circular fibres of the superior constrictor muscles.

The most important muscles of the sphincter are the levators and the palatopharyngeus muscles.

The levators are strong fleshy muscles, arising from the base of the skull medial to the cartilagenous part of the eustachian tube, and from the medial wall of the eustachian cartilage itself. Each levator muscle winds under the lower edge of the medial wall of this cartilage, and passes below the tube, its muscle mass giving rise to a prominence in the floor of the tube. Viewed from the nasopharynx, with the soft palate depressed, the muscle is seen to sweep downwards to the upper surface of the palate to fuse with its fellow of the opposite side. The action of the levators is to pull up the soft palate so that it closes off the nasopharynx completely (Fig. 3).

The levators are the main sphincters of the nasopharynx, and as they contract they pull up the twin palatopharyngeus muscles, which in turn pull up the circular fibres of the superior constrictors. The contraction and relaxation of these muscles helps to drain secretions in the nasopharynx into the pharynx, by suction action.

The Opening and Closing of the Eustachian Tube is brought about mainly by the contraction and relaxation of the levator muscles, with the assistance of the tensors and salpingopharyngeus muscles. When the levators contract, their muscle masses rise in the opening of the tube filling its lumen firmly. At the point where the levators

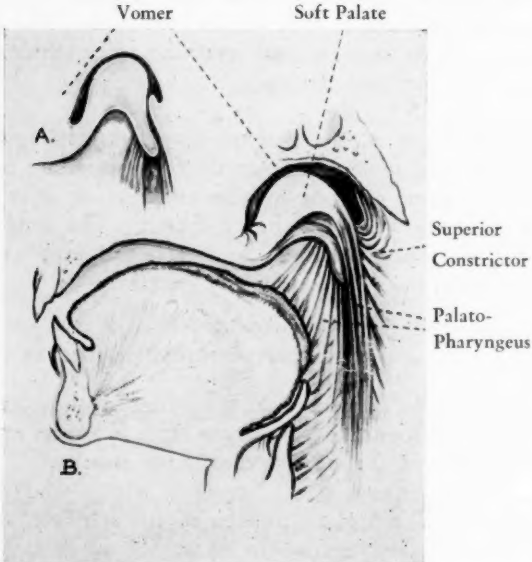


Figure 3

relax and the soft palate is pulled down by the tensors, the valve-like slit in the anterior part of the eustachian tube opens momentarily. It is probable that suction action brought about by the contraction and relaxation of the levators helps to drain the eustachian tube of its mucus secretions.

The palatopharyngeus muscles are paired muscles forming, during relaxation of the pharynx, the posterior pillars of the pharynx. Below, their fibres fan out over the lateral and posterior walls of the pharynx. Above, their fibres divide into two bundles on each side and are attached to the posterior surface of the soft palate, enclosing the posterior fibres of the levators.

On contraction of the levators during extreme elevation of the palate, with maximum closure of the nasopharyngeal sphincter, the palatopharyngeus muscles are pulled upwards so that they meet in the middle line. In turn they pull up the superior constrictor circular fibres causing a ridge to be formed on the posterior wall of the nasopharynx (Fig. 4).

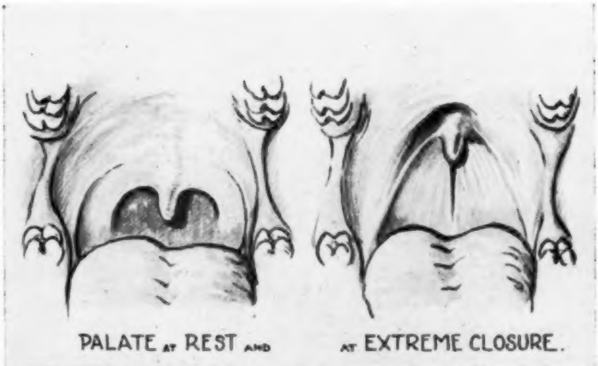


Fig. 4.—Action of palato-pharyngeus in closure of the naso-pharyngeal sphincter—extreme.

It will be seen that both the paired levators and the paired palatopharyngeus muscles are attached to the upper and posterior aspects of the soft palate, and together form the upper-posterior muscle layer of the palate. The central muscle layer is formed by the fan-like aponeurosis of the paired tensors. The lower-anterior muscle layer of the palate is formed by the paired palatoglossus muscles (Fig. 5).

Anything (like scarring or trauma) that limits free movement of the palatopharyngeus muscles of the palate (posterior pillar of the fauces) must therefore limit movements of the levators. This is very important.

Each of the tensors of the palate is arranged rather like a double fan of muscle fibres with a central pulley-like tendon which runs over the hamulus of the pterygoid process. The upper half of each tensor muscle is attached to the base of the skull lateral to the cartilagenous eustachian tube, and to the lateral wall of the eustachian cartilage. The fibres narrow to the tendon, pass over the hamulus, and then fan out again to join the muscle of the opposite side. Anteriorly the muscle is inserted into the edge of the hard palate. Centrally it divides to enclose the musculus uvulae (Fig. 5).

The tensor muscles of the palate must function during sleep to prevent flapping with each respiratory interchange, and during heavy

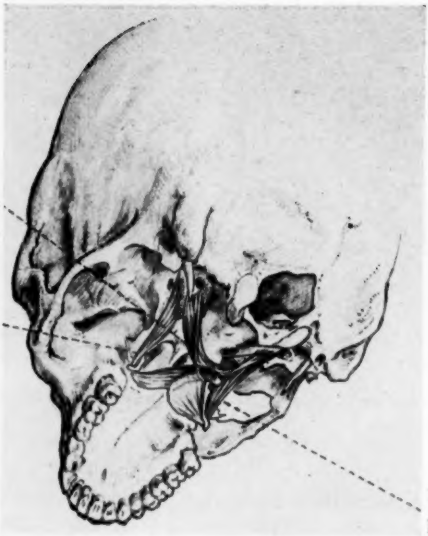


Figure 5

exercise, when both primary (nasal) and secondary (mouth) airways are used. The tensors are the platform-fixing muscles, which fix and hold firm the central layer of the soft palate. The degree of tenseness varies. Relaxation of the tensors allows full contraction of the levators and full elevation of the palatopharyngeus muscles when the nasopharyngeal sphincter is closed. During the initiation of the second involuntary stage of swallowing, both the nasopharyngeal sphincter and the mouth sphincter have to close. The tensors must therefore strike a balance between the actions of the levators and palatopharyngeus muscles on the one hand, and the palatoglossus muscles on the other, and in fixing the plane of the palate in differing positions, to allow the various muscle patterns to perform their required functions.

The musculus uvulae is attached to the posterior spine of the hard palate and, when it contracts, it shortens the soft palate and helps to fill the sphincter formed by the two palatopharyngeus muscles (Figs. 4 and 5).

The function of the soft palate group of muscles is therefore very complex.

1. They close off the nasopharynx from the pharynx with strong sphincter-like action (levators and palatopharyngeus muscles).

2. They help in closing the mouth sphincter in the anterior wall of the pharynx (palatoglossus muscles).

3. They act as a hinged plane attached to the posteriar bony edge of the hard palate, which can be fixed and held rigidly in certain planes to assist respiration.

4. Their sphincteric action helps to drain the nasopharynx of mucus secretions (levators and palatopharyngeus muscles).

5. Their sphincteric action, of closure and relaxation, opens and closes the eustachian tube, and assists drainage of mucus from the tube (levators and tensors).

Partial paralysis, or even subclinical paralysis of the soft palate sphincter, or pseudoparalysis due to fixation of the soft palate muscles by scar tissue after tonsillectomy or other trauma, or interference with the sphincter due to partial cleft palate, or extreme hyperplasia of tonsils buried in the palate muscles and limiting free movement of the palate, or hyperplasia of adenoid tissue limiting sphincteric closure, must interfere greatly with function.

Problems that can arise from this interference with function are:

1. Inefficient drainage of dust-laden mucus from the nasal passages into the pharynx, so contributing to nasopharyngeal stasis, infection, and postnasal catarrh.

2. Interference with adequate opening and closing of the eustachian tubes leading to middle ear deafness.

3. Interference with palatal speech, particularly in the very young during development of speech.

4. Inefficient closure of the nasopharynx during swallowing, or vomiting, leading to occasional passage of fluids or foods into the nasopharynx.

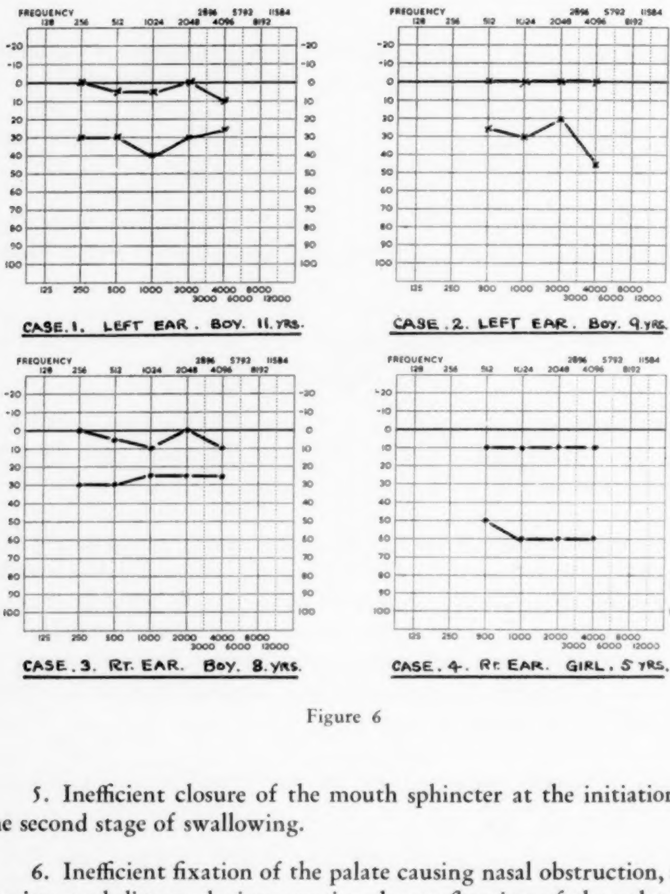


Figure 6

5. Inefficient closure of the mouth sphincter at the initiation of the second stage of swallowing.

6. Inefficient fixation of the palate causing nasal obstruction, and snoring, and distress during exertion due to flapping of the palate on deep respiration.

Hearing may be completely restored in children, or greatly improved, by freeing muscles of the soft palate caught up in scar tissue after tonsillectomy, showing how limitation of movement and function of the sphincter causes the deafness (Fig. 6). If these muscles control such important functions, then every effort should be made to prevent damage to them during the operation of tonsillectomy, and during operations for the removal of adenoid tissue from the nasopharynx.

Scarring of the soft palate muscles, or even destruction of these muscles, is unfortunately seen too often. It is only by understanding the functions of these important muscle groups more fully that we shall learn to respect and protect them. Careful removal of tonsils and adenoid tissue need not cause scarring and limitation of function of the nasopharyngeal sphincter. Indeed it is common knowledge that considerable improvement in hearing, nasopharyngeal catarrh, and nasal obstruction, follows careful removal of hyperplastic tonsil and adenoid tissue which itself had been limiting proper movement of the soft palate muscles.

COMPENSATION

In bulbar paralysis (nucleus ambiguus) it is usually the levator palati, palatopharyngeus, and palatoglossus of one side, and the uvula, that is partially or wholly paralyzed.

The tensor is supplied by the V nerve via the otic ganglion. If its motor nucleus is also involved, then the soft palate will be useless and adhere to tongue, or posterior pharyngeal wall, and cause distress and grave interference with the functions of respiration, swallowing and speech.

The usual residual form of pharyngeal paralysis after poliomyelitis is a one-sided paralysis of the soft palate, larynx, and pharynx, including the lower sphincter of the pharynx. There is an asymmetrical contraction of the soft palate to the opposite side, loss of movement in one vocal cord, and pooling of mucus in the pyriform fossae at the opening of the esophagus.

Compensation is seen in the efforts of the intact soft palate muscles to contract and close the partly paralyzed nasopharyngeal sphincter; in the efforts of the three constrictor muscles of one side to propel the food down into the esophagus; in the intact half of the lower pharyngeal sphincter to overcome the handicap of the paralysis of its other half (palatopharyngeus); in the efforts of the intact muscles of the laryngeal inlet to close the laryngeal sphincter when one-half is paralyzed, and to open it for respiration, and to move it during speech. The intact muscles of the partially paralyzed nasopharyngeal sphincter will try to overcome the inaction of the paralyzed side in movements required for speech, and in raising the tongue at the beginning of the first stage of deglutition (palatoglossus).

During respiration the nonparalyzed muscles will endeavor to pull the partly paralyzed palate away from the posterior pharyngeal wall to allow nasal breathing, the tensor helping greatly if the V nerve nucleus has escaped.

UNDIAGNOSED PARALYSIS OF THE PHARYNX (30 YEARS)

CASE 1. Housewife, aged 47. This patient first attended hospital complaining of a dry mouth, from mouth breathing at night. Her family doctor sent a note suggesting that her deflected nasal septum might be the cause. She was a pleasant, uncomplaining type of woman.

On examination she was found to have a paralysis of the left side of her soft palate, a paralyzed left vocal cord, and delay in emptying the lower pharynx into the esophagus, as shown by collected frothy mucus and saliva in both pyriform fossae.

She had no knowledge whatever of any paralysis of her pharynx, nor had any doctor ever noted it.

On investigation she remembered having had poliomyelitis 30 years previously, at the age of 16, but it was thought that she had made a complete recovery. On questioning, she admitted to postnasal catarrh, with occasional regurgitation of fluids into the nose; sometimes she had had difficulty with the enunciation of words with palatal sounds. Her voice had tended to tire after speaking for any length of time, and sometimes it had been an effort to talk, but she was not aware that anything was wrong with her larynx. Since she was 16, she had always found it rather a struggle to swallow things like mashed potato. She said that if she took a good drink "it would go down."

Compensation had been extremely good, in spite of the fact that one side of the soft palate, pharynx and larynx had been paralyzed for 30 years. The primary functions of the pharynx, a) to maintain a satisfactory airway for respiration, b) to function adequately in the second (involuntary) stage of swallowing, c) to allow clear speech (pharyngeal muscles, as well as tongue and larynx), had been very efficiently preserved. Only when she contracted a head cold (upper respiratory infection) in the winter did she experience difficulty. Then her cough was troublesome. She said that sometimes she found

it difficult to laugh. There were times when she found it difficult to clear her throat when talking.

She was greatly reassured when told the cause of her difficulties, and her home life was made easier because those living with her no longer regarded the clearing of her throat, and her idiosyncracies in swallowing, as irritating habits. They, and she, realized that there were physical causes for her symptoms. This knowledge proved to be of the greatest help to her. She was also told how to use postural drainage (knee-elbow position) if she felt she was choking because of the overflow of saliva and mucus into the partially paralyzed larynx during upper respiratory infections. This, too, gave her much reassurance.

Compensation was clearly demonstrated in the closure of the nasopharyngeal sphincter by overaction of the nonparalyzed side, in the emptying of the pharynx into the esophagus with the nonparalyzed constrictors, and in the right vocal cord coming right across the midline to close off the larynx in adduction.

The unilateral palatal paralysis was responsible for

1. The postnasal catarrh, caused by the inefficient suction action of the levator palati muscles in draining the nasopharynx of mucus.

2. The difficulty with palatal speech sounds, particularly "L" and "R".

3. The occasional regurgitation of fluids into the nose on swallowing. This was due to occasional inefficient closure of the nasopharynx because half the sphincted muscles were paralyzed. Palatopharyngeus and levator palati being chiefly at fault. The remarkable thing is that this regurgitation so seldom took place.

4. Some, at any rate, of the hearing loss was due to inefficient opening and shutting of the eustachian tubes on swallowing. The levator palati being chiefly at fault, but also the salpingopharyngeus. Some of the deafness was undoubtedly due to scarring and partial fixation of the ossicles from old otitis media, and this might well have been secondary to 1) the postnasal catarrh caused by the inefficient drainage of the nasopharynx and 2) inefficient opening and shutting of the eustachian tube.

The left-sided paralysis of the pharyngeal constrictors was responsible for

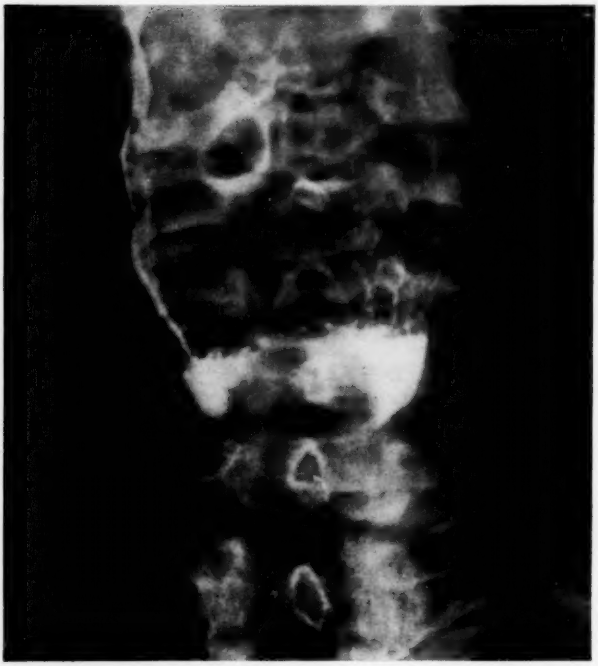


Figure 7

1. The difficulty in swallowing food, particularly solids. The left cricopharyngeus was paralyzed, and in spite of excellent compensation by the nonparalyzed side, frothy mucus and saliva tended to collect in the pyriform fossae, and to overflow into the partially paralyzed larynx on occasion, causing an irritating cough.

2. This also accounted for the desire to clear the throat. The effort to do this being of itself somewhat less efficient than normal, because of the partial laryngeal paralysis. It was this effort to clear the throat when eating, drinking, and speaking, that was one of the sources of greatest annoyance to other people.

The left-sided laryngeal paralysis caused:

1. The voice to tire easily, because of the overaction of the nonparalyzed cord. Whilst the larynx was at rest, the paralyzed cord lay inert, just lateral to the midline, with its corniculate cartilage

pointing posteriorly, and its arytenoid cartilage lying just anterior to the nonparalyzed arytenoid. On adduction, the nonparalyzed arytenoid travelled across anteriorly to the paralyzed arytenoid. This tendency of the nonparalyzed arytenoid to lie just posterior to the paralyzed arytenoid during relaxed breathing is a great help in diagnosis of any hemiparalysis of the larynx, should laryngoscopy, for any other reason, prove difficult. It is important to note that there is often some slight movement of the paralyzed arytenoid in unilateral paralysis of the larynx. This is due to the pull of the nonparalyzed half of the transversus arytenoid muscle. Some movement may be due to motor fibres in the internal branch of the superior (mainly sensory) laryngeal nerve that have escaped when the recurrent laryngeal nerve was paralyzed.

2. The occasional inefficient cough (ptussive reflex) due to the impaired sphincter action of the larynx.

The respiratory airway remained good. The carbonic acid content of the blood, controlling the medullary respiratory centers, caused sufficient abduction of the nonparalyzed cord to allow efficient oxygenation. The cords of a normal larynx abduct just a little with every inspiration, and adduct a little with each expiration. During cyanosis the cords of a normally intact larynx are held in greater abduction automatically, and the slight abductor movements on inspiration, and adductor movements on expiration are maintained with a wider larynx. During any stress imposing oxygen lack, the nonparalyzed cord would have to abduct more widely than normal to compensate for the absence of movement on the paralyzed side. She found a little discomfort on exertion. She described difficulty in laughing. The rather excessive, spasmodic movements of the pharynx in laughing would place greater strain on compensation by the nonparalyzed side.

This case was an excellent example of compensation shown by the intact pharyngeal muscles over a period of 30 years. An x-ray taken during swallowing showed the delay in emptying the pharynx. Using only a very little diluted liquid barium, this was seen to collect in both pyriform fossae and in both valleculae at each side of the epiglottis. These confirmatory diagnostic signs are seen also in high esophageal obstruction, or postcricoid obstruction from other causes, like carcinoma. The signs indicate delay in emptying the pharynx. Very little diluted barium must be used, or too much opaque barium will obscure the picture, and also may overflow into the larynx causing needless distress (Fig. 7).

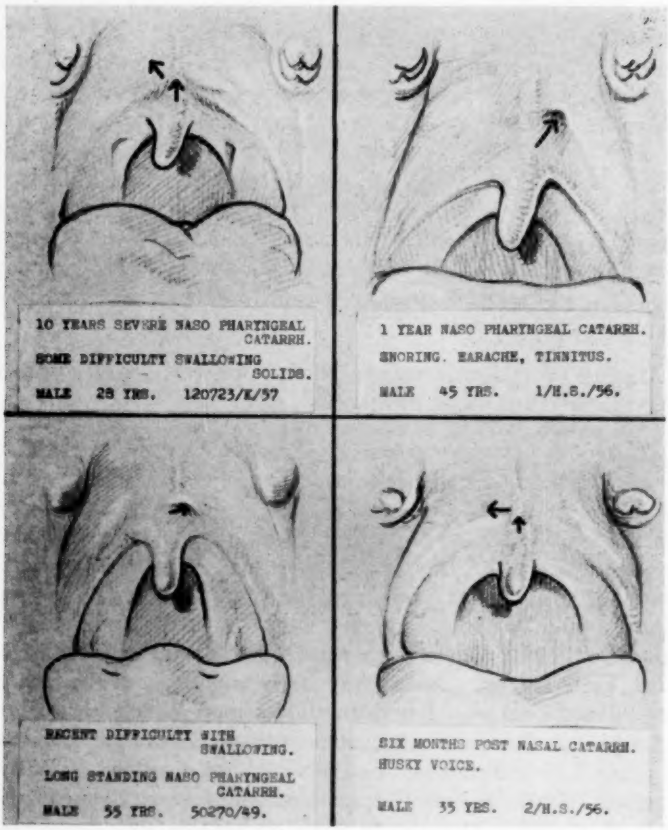


Figure 8

UNDIAGNOSED PARALYSIS OF THE PHARYNX (14 DAYS)

CASE 2. Married man, aged 39 years. Attended first in 1955, he had had a febrile illness 14 days previously, which was diagnosed as tonsillitis, and treated with penicillin. He said he now felt better in himself, but for the past week had had difficulty in swallowing solids, no pain, but his voice had been a little husky.

On examination he had a paralyzed left side of the soft palate, frothy mucus collected in both pyriform fossae to denote delay in

emptying the pharynx into the esophagus, and a paralyzed left side of the larynx. This is rather a typical triad of signs in pharyngeal paralysis, where the nucleus ambiguus on only one side of the medulla has been injured. The large motor cells in one nucleus ambiguus supplying the cranial part of the spinal accessory nerve, are affected. Their motor fibres travel in the vagus (joining the vagus at the inferior ganglion) in the pharyngeal branch, external branch of the superior laryngeal, and in the recurrent laryngeal branch. The importance of this particular case lies in the fact that the paralysis was entirely unsuspected by both patient and doctor, and might quite easily have remained unsuspected, like the former case. There was no limb weakness, and no other evidence of paralysis.

We saw the wife and man together and explained what had happened. This gave both much relief and understanding as time went on. He was told to swallow deliberately and to take his time. He was told to use drinks to aid swallowing. His wife was told the reason why he would make irritating noises to clear his throat. He was warned that if he contracted a head cold, or upper respiratory infection, he might feel he would choke on waking from sleep, and so was told how to use postural drainage in the knee-elbow position on his bed, so that he could more easily get rid of mucus and saliva overflowing into his larynx. This was only an emergency measure, intended to give the patient confidence. Both the patient and his wife were reassured that he would be seen at intervals, so that observation could be kept for signs of recovery in paralyzed muscles.

He was last seen in March 1957 (one year and eight months after the onset of his "tonsillitis"). He still had "phlegm in his throat" and found it tiring to clear it. He still had to swallow deliberately, and take his time. His voice had improved, but still tended to tire, and remained somewhat hoarse. He still had to clear his voice a good deal.

On examination, the left side of the soft palate and the left side of the larynx were still paralyzed. No change could be detected. The left arytenoid still lay anteriorly to the unparalyzed arytenoid when the larynx was relaxed; the nonparalyzed arytenoid still passed anteriorly to the paralyzed arytenoid on adduction. The collection of frothy mucus in the pyriform fossae had disappeared, showing some probable recovery in the pharyngeal constrictors, or very efficient compensation by the nonparalyzed side. The patient said that he had ceased to cough on drinking. Presumably there had now ceased to be

overflow into the partially paralyzed larynx on swallowing. He could now manage "nearly a cup of tea without a pause, but not quite."

Speech was good, but he had a little difficulty sometimes with "L's" and "R's". Sneezing was rather alarming to him. "The whole throat seems to come up, and I feel I can't get it back again."

FOUR EXAMPLES OF UNDIAGNOSED PALATAL WEAKNESS FOUND ON ROUTINE EXAMINATION (FIG. 8)

CASE 1. Male, 28. Symptoms: Postnasal catarrh. Some difficulty in swallowing solids. Examination: Slight left palate weakness. The levator and tensor on the left appeared to be weak. No froth collected at opening of esophagus. No laryngeal weakness detected.

CASE 2. Male, 35. Symptoms: One year tinnitus, slight earache, postnasal catarrh, snoring at night. Examination: Slight persisting weakness right side of palate. No signs of other pharyngeal or laryngeal weakness seen.

CASE 3. Male, 55. Symptoms: Long-standing nasopharyngeal catarrh, some difficulty with swallowing. Examination: Slight weakness palate muscles on the right side. No pharyngeal or laryngeal weakness detected.

CASE 4. Male, 35. Symptoms: Six months postnasal catarrh, husky voice. Examination: Palate rises symmetrically, and is then pulled over to the right, suggesting weakness of the left palate muscles, particularly of the tensor.

FOUR EXAMPLES OF PHARYNGEAL WEAKNESS. PALATE AND PHARYNX OR LARYNX. UNDIAGNOSED. (FIG. 9)

CASE 1. Male, 20. Symptoms: Four years, postnasal catarrh. Examination: Left palate weakness, slight weakness of left vocal cord. No froth at the opening of the esophagus.

CASE 2. Male, 55. Symptoms: Postnasal catarrh, and inability to get rid of mucus from pharynx. Examination: Right palate weakness, froth right pyriform fossa to suggest delay in emptying the pharynx.

CASE 3. Male, 33. Symptoms: One year, recurrent sore throats. Examination: Slight left palate weakness, bifid uvula. Diphtheria aged 3.

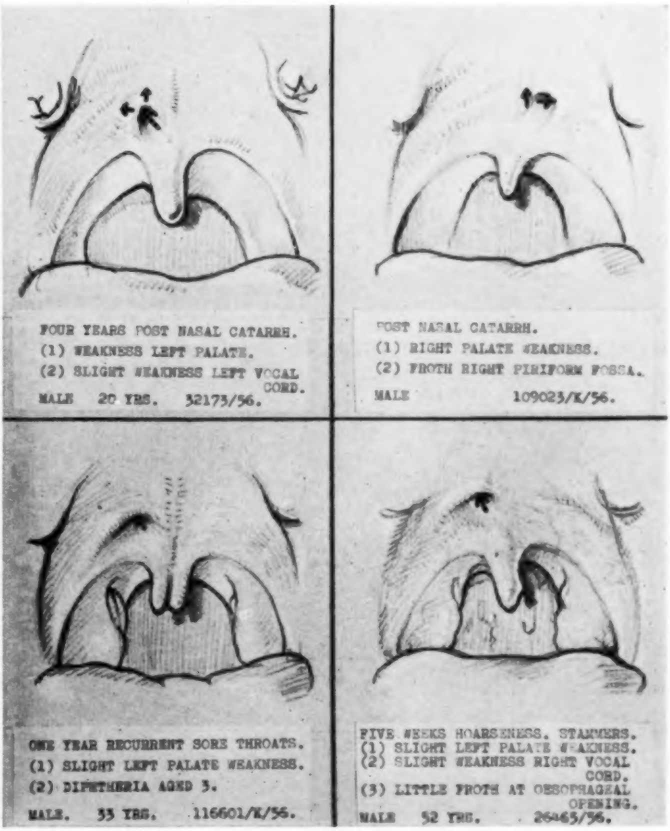


Figure 9

CASE 4. Male, 52. Symptoms: Five weeks, hoarseness. Used to stammer. Examination: Slight left palate weakness, slight weakness of right vocal cord, a little froth collected in pyriform fossae.

FOUR EXAMPLES OF SCARRING OF SOFT PALATE. (FIG. 10)

CASE 1. Schoolmistress, 28. Absence of both palatoglossal folds. Tonsillectomy in childhood. Symptoms: Postnasal catarrh, pharyngitis and bronchitis.

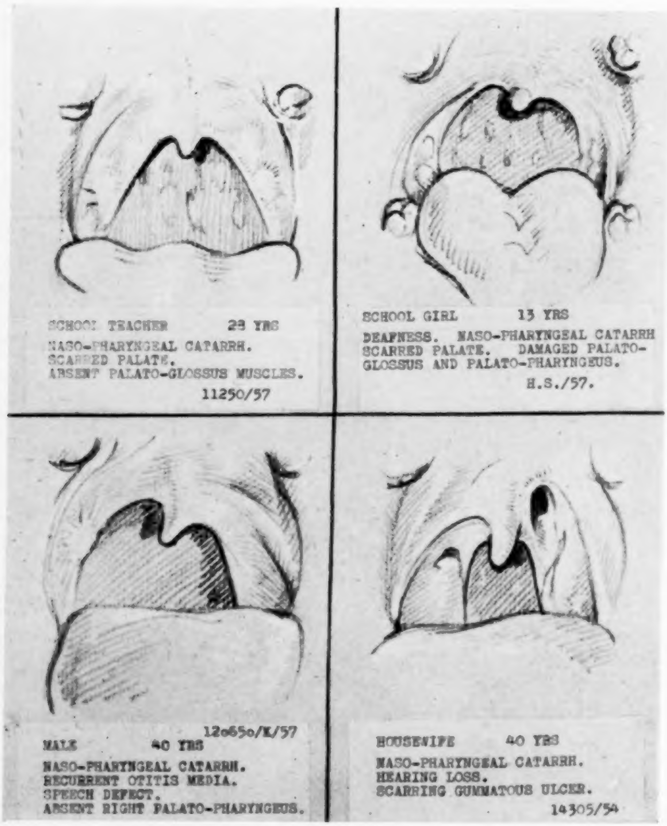


Figure 10

CASE 2. Schoolgirl. Absence of part of palatoglossus, palatopharyngeus, both sides. Tonsillectomy 1949. Symptoms: Postnasal catarrh, pharyngitis, hearing loss.

CASE 3. Male, 40. Absence of palatopharyngeus, right. Tonsillectomy in childhood. Symptoms: Postnasal catarrh, recurrent otitis media, some hearing loss, difficulty with palatal speech.

CASE 4. Housewife, 40. Severe scarring from gummatous ulceration of palate. Symptoms: Postnasal catarrh, hearing loss.

Patients with scarring due to gummata seem to complain very little, in spite of severe scarring and destruction.

FOUR CASES SHOWING IMPROVEMENT IN HEARING AFTER FREEING OF PALATE MUSCLES. (FIG. 6) PSEUDOPARALYSIS - DEAFNESS

CASE 1. *Scarred Palate.* Schoolboy, aged 11 years. Severe deafness at school interfering with education. Palatopharyngeus and palatoglossus caught up in scar tissue. Tonsillectomy elsewhere 1952. Hearing loss: R. db. 20/30; L. db. 25/40. Freeing of palate muscles 11-11-55. Hearing after: R. db. 0/5; L. db. 0/10.

Schoolmaster, family doctor, school medical officer, all reported great improvement in ability after hearing improved. Inattention and irritation were the result of straining to listen.

CASE 2. *Old Diphtheritic Paralysis of Palate with Scarring of Palate.* Schoolboy, aged 9 years. Tonsillectomy 1949. Right-sided diphtheritic paralysis of palate 1953. Deafness, postnasal catarrh, earache, tinnitus for six years. Hearing loss 21-4-55. R. db. 0/20; L. db. 20/40. Freeing of palatopharyngeus muscles 13-12-55. Hearing loss 7-3-57. R. db. 0/10; L. db. 0/0.

This shows good recovery of hearing with relief from earache, tinnitus, and postnasal catarrh after freeing of palate muscles.

CASE 3. *Scarred Palate Limiting Movement of Palatopharyngeus, Levators, and Tensors.* Schoolboy, aged 8 years. Opening and closing of eustachian tubes interfered with because palatopharyngeus was held down by scar tissue. Levators could not lift up the palate properly. Drainage of nasopharynx was interfered with. He had had an operation on his tonsils at the age of 4½ years.

Hearing, 1954: average R. 25/30 db.; L. 20/45 db. Freeing of palate muscles 26-7-55. Hearing after, 1957: R. 0/10 db.; L. 5/10 db.

CASE 4. *Hyperplasia of Tonsil and Adenoid Tissue Limiting Palatal Movement. Tonsils Buried in Soft Palate.* Girl, aged 5 years. Attended 1957 with deafness, postnasal catarrh, palatal speech "L", "R". Hearing 31-1-57: 50/55 db. loss. Recovered to 10 db. loss three days after freeing palate muscles, and maintained. The levators and tensors could not act fully, and so could not produce adequate opening and closing of eustachian tube.

SUMMARY

Common complaints like nasopharyngeal catarrh, difficulties with swallowing, and clearing the throat, snoring, mouth breathing, difficulties with speech, and conductive deafness can be caused by 1) undiagnosed, but clinically demonstrable, pharyngeal paralysis (poliomyelitis or other virus medullary polyneuritis), 2) weakness of pharyngeal muscles with no true clinical evidence of paralysis (after diphtheria or poliomyelitis), that is, subclinical paralysis, 3) pseudoparalysis of the pharynx from a) scarring of the palate following tonsillectomy, or other trauma, b) hyperplasia of tonsil and adenoid tissue, c) clefts of the soft palate.

Further information on the anatomy and function of the soft palate muscles is presented, particularly in relation to the mechanisms of draining the nasopharynx, and of opening and closing the eustachian tubes. The importance of respecting these very important muscles during the operations of tonsillectomy, and removal of adenoids, is stressed.

Examples of undiagnosed pharyngeal paralysis are described. Improvement in hearing after release of scarred palate muscles is demonstrated. The neurology and function of the pharynx is discussed.

The author therefore offers this conclusion:

1) If partial paralysis of the pharyngeal muscles can be present without anyone knowing about it (undiagnosed paralysis),

2) If weakness of the pharyngeal muscles can be present without any clinical signs (subclinical paralysis), and

3) If we appreciate the important function of the palate muscles, then,

4) If we can prevent scarring of the palate muscles (pseudoparalysis), we shall prevent very many children from growing up with nasopharyngeal catarrh and deafness in later life.

BIBLIOGRAPHY

Browne, Denis: The Operation for Cleft Palate. Brit. J. Surg. 20:7, 1932.

Cunningham's Text-Book of Anatomy, 9th edition, edited by J. C. Brash, London: Oxford University Press, p. 444, 1951.

Gray's Anatomy, Descriptive and Applied, 30th edition, edited by T. B. Johnston and J. Whillis, London: Longmans, Green & Company, pp. 1270, 1335, 1338, 1339; 1949.

Keogh, Charles: Paralysis of the Pharynx. J. Laryng. 70:344, 1956.

Last, R. J.: Anatomy, Regional and Applied. London: Churchill, pp. 447, 448, 476, 481, 489; 1954.

Lemere, F.: Innervation of Larynx; Innervation of Laryngeal Muscles. Amer. J. Anat. 51:417, 1932.

Negus, V. E.: The Comparative Anatomy and Physiology of the Larynx. London: Heinemann, pp. 94, 161, 1949.

Sharrard, W. J. W.: Correlation Between Changes in the Spinal Cord and Muscle Paralysis in Poliomyelitis—A Preliminary Report. Proc. R. Soc. Med. 46:346, 1953.

Whillis, J.: A Note on the Muscles of the Palate and the Superior Constrictor. J. Anat. Lond. 65:92, 1930.

Wood Jones, Frederic: The Nature of the Soft Palate. Ibid. 74:147, 1940.

XXXIII

THE GONIOMETER TEST

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DATA FROM 280 NORMALS

Of the several functional tests regarding the human equilibrium as a whole, the goniometer test (a tilt test) is considered to be one of the most important clinically.

The evolution of this test really began in 1892 when Stein[1] reported his first work in a meeting at the International Society in Moscow. He paid most attention to the angle of fall, and for open eyes of normal human subjects found it to vary from 39° to 18°. Thereafter methods of advancing technique and clinical use of such tests have been sought by many authors notably Watusji,[1] Kubo[2] and Kosogabe, and a number of investigators reported the results obtained by their own methods (Ikeda,[1] Takasaki and his associate[2] and Kumagai,[3] etc.).

Judging by the reports of recent years, one can conclude that the results obtained by the methods heretofore in use show a large individual variation in normal persons and a wide range even in the same subject at different times. It is, therefore, difficult to distinguish between normal and pathological data. Actually things have come to such a state that some investigators would rather do away all together with the goniometer test.

The aims of the present investigation are to study methods with the aid of a fairly comprehensive set of normal data, and to attempt to discover sources of error and, moreover, to improve on the method.

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Read at the Sixth International Congress of Otolaryngology, Washington, D. C., May 1957.

The vestibular organs were stimulated during the tests by continuously variable speeds of inclination, since the equipment employed by the above mentioned investigators was a hand-machine.

To remedy this defect electrically driven equipment, or the goniometer, was devised by S. Honjo.

This paper presents normal data tested by the author's equipment.

MATERIAL AND METHODS

For test subjects to provide normal data we used 280 healthy adults (16 to 60 years of age) of whom 230 were male and 50 female, all having normal audiograms and negative otoneurological histories.

The equipment devised by S. Honjo was used (Fig. 1). The detailed description of this apparatus will be given in a separate paper by the author. Plank (p) moved by electric driving gears (g) is inclined at six speeds of inclination, 1°, 3°, 5°, 10°, 15°, and 20° (Fig. 1).

From the normal erect posture on the horizontal plank of the goniometer each subject was inclined forward, backward and to both sides, at six different speeds of inclination. During the inclination the subject kept his eyes open or closed.

As "angle of fall," we recorded the angle at which the subject fell from the plank.

The subject was to maintain similar conditions in each test as far as possible; for instance, shoes were removed and each subject wore the same type of stockings. In principle there was an interval of at least five minutes between each test in different directions.

RESULTS

Table I gives the mean and the range of fall angles with open eyes on 280 normal persons, who have been inclined in four directions (anteriorly, posteriorly and to both sides) at different six speeds of the inclination.

At the inclination speed of 1°/sec. the mean of fall angle shows the smallest value (the mean of fall angles in four directions is 29.89°). As the inclination speed increases, so the mean of the fall angle gains gradually, so that the inclination speed of 20°/sec. shows the largest

TABLE I

MEAN AND RANGE OF FALL ANGLE IN FOUR DIRECTIONS AT VARIOUS INCLINATION SPEEDS WITH OPEN EYES

INCLINATION SPEED PER SEC.	NO. OF CASES	ANTERIOR M ± S.E.	S	POSTERIOR M ± S.E.	S	RIGHT M ± S.E.	S	LEFT M ± S.E.	S
1°	280	30.15 ± 0.14 (37-25)	2.400	29.65 ± 0.14 (38-24)	2.339	30.15 ± 0.14 (36-24)	2.364	29.61 ± 0.12 (36-24)	2.173
3°	280	32.32 ± 0.14 (38-27)	2.341	31.91 ± 0.14 (38-26)	2.341	32.33 ± 0.14 (38-27)	2.406	31.66 ± 0.12 (37-27)	2.012
5°	280	34.67 ± 0.15 (42-29)	2.470	34.03 ± 0.14 (41-28)	2.402	34.43 ± 0.14 (40-28)	2.406	33.98 ± 0.14 (41-28)	2.280
10°	280	38.28 ± 0.15 (45-32)	2.553	37.89 ± 0.14 (44-31)	2.377	38.28 ± 0.14 (46-32)	2.319	37.87 ± 0.14 (46-32)	2.324
15°	280	41.21 ± 0.16 (48-35)	2.668	40.96 ± 0.16 (48-34)	2.663	41.14 ± 0.15 (48-35)	2.472	40.44 ± 0.14 (47-35)	2.302
20°	280	44.30 ± 0.16 (52-38)	2.737	43.95 ± 0.16 (51-37)	2.760	43.81 ± 0.16 (51-37)	2.707	43.34 ± 0.14 (50-38)	2.406

TABLE II

MEAN AND RANGE OF FALL ANGLE IN FOUR DIRECTIONS AT VARIOUS INCLINATION SPEEDS WITH CLOSED EYES

INCLINATION SPEED PER SEC.	NO. OF CASES	ANTERIOR M ± S.E.	ANTERIOR S	POSTERIOR M ± S.E.	POSTERIOR S	RIGHT M ± S.E.	RIGHT S	LEFT M ± S.E.	LEFT S
1°	280	29.61 ± 0.15 (36-24)	2.580	28.65 ± 0.13 (37-23)	2.315	29.46 ± 0.15 (35-24)	2.498	28.92 ± 0.13 (36-23)	2.261
3°	280	31.56 ± 0.14 (38-27)	2.398	31.09 ± 0.14 (38-26)	2.373	31.62 ± 0.14 (38-27)	2.335	30.92 ± 0.12 (38-27)	1.942
5°	280	33.93 ± 0.16 (42-28)	2.600	33.29 ± 0.14 (42-28)	2.371	33.87 ± 0.15 (40-28)	2.492	33.46 ± 0.13 (40-28)	2.184
10°	280	37.90 ± 0.15 (45-32)	2.439	37.47 ± 0.15 (45-30)	2.445	37.95 ± 0.14 (45-32)	2.264	37.50 ± 0.14 (46-32)	2.396
15°	280	40.74 ± 0.16 (49-35)	2.644	40.54 ± 0.14 (47-35)	2.280	40.86 ± 0.15 (47-34)	2.534	40.34 ± 0.14 (49-34)	2.352
20°	280	43.70 ± 0.17 (52-37)	2.864	43.43 ± 0.16 (51-36)	2.726	43.53 ± 0.15 (51-37)	2.561	43.04 ± 0.15 (51-37)	2.506

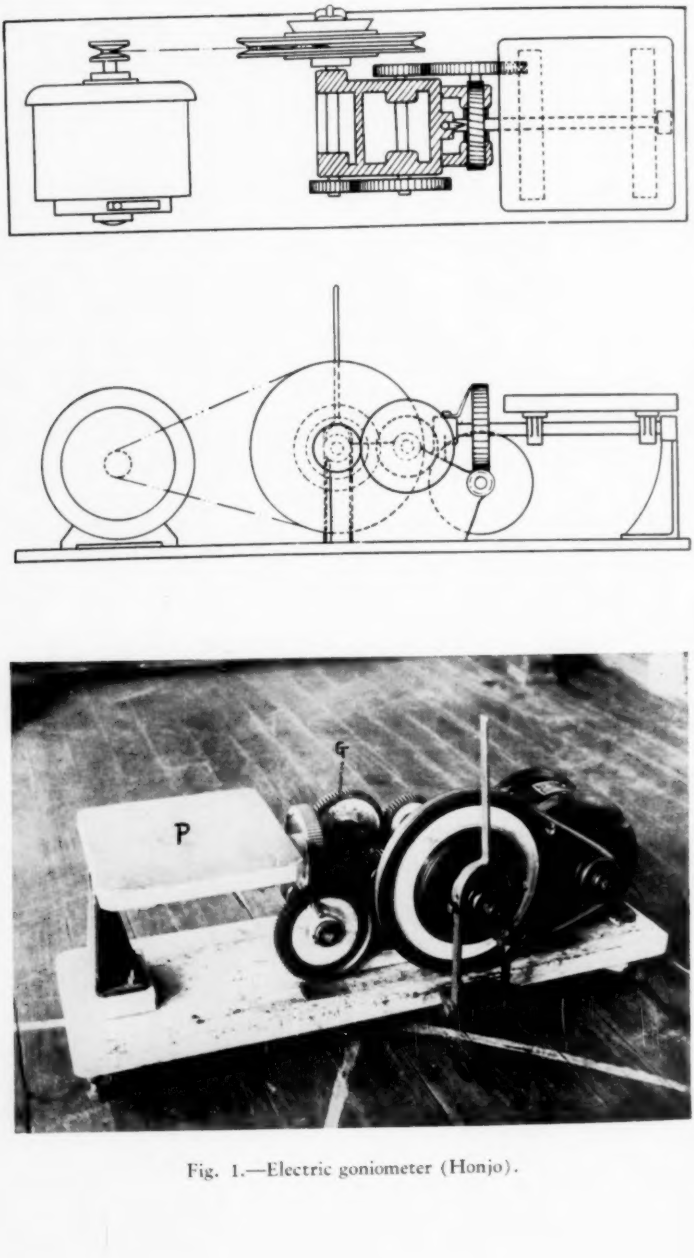


Fig. 1.—Electric goniometer (Honjo).

figure, the mean of fall angles in four directions being 43.85°. It is interesting to note that with increasing inclination speed there is a progressive increase of the fall angle. The fall angle in each of four directions is directly proportional to the speed of inclination.

The graphical equivalents of Table I clearly show that under the aforementioned conditions the mean of fall angles can be represented as the following linear function:

$$Y = aX + b$$

where a is the coefficient of direction and b is the mean of fall angles.

$$\text{Therefore} \quad Y = 0.824\ X + 29.89$$

Table II shows the mean and range of fall angles with closed eyes on 280 normal persons, who have been inclined in four directions at six speeds with eyes kept open. The mean of fall angle is the smallest at the inclination speed of 1°/sec., the mean of fall angle in four directions being 29.16°, increasing with an increased inclination speed of 20°/sec. The curve of the fall angle with closed eyes runs graphically parallel to those of open eyes (Fig. 2), as shown in the following linear function:

$$Y = 0.844\ X + 29.16$$

As concerns the difference between each fall angle to four directions at the same inclination speed, every value is less than 1° (Table I and II).

If the readings differing less than 1° are accepted as equal, the following conclusion may be obtained:

At the same inclination speed the fall angle is not influenced by the direction of inclination in a normal person.

Table III represents the individual variations (= max — min) of fall angle on 280 normal persons. The greatest variation is 15° and the smallest 10°. Generally speaking, the variations at the lower speeds (1° to 3°/sec.) are smaller than at the higher speeds (15° to 20°/sec.).

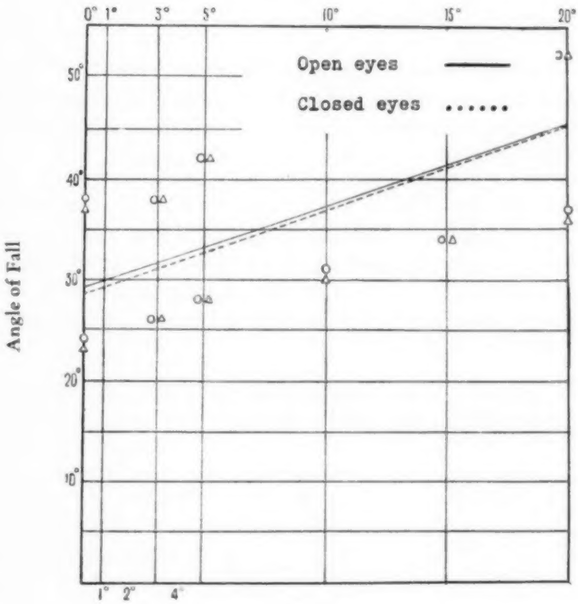
It is remarkable that the physiological individual variation of the fall angle lies in the range of 15° to 10°.

A comparison of the figures obtained with open and with closed eyes at the same inclination speed shows that, by the methods here

TABLE III

VARIATIONS (= MAX. — MIN.) OF FALL ANGLE IN FOUR DIRECTIONS AT VARIOUS INCLINATION SPEEDS ON 280 NORMAL PERSONS

INCLINATION SPEED PER SEC.	NO. OF CASES	ANTERIOR		POSTERIOR		RIGHT		LEFT	
		OPEN EYES	CLOSED EYES	OPEN EYES	CLOSED EYES	OPEN EYES	CLOSED EYES	OPEN EYES	CLOSED EYES
1°	280	12°	12°	14°	14°	12°	11°	12°	13°
3°	280	11°	11°	12°	12°	11°	11°	10°	11°
5°	280	13°	14°	13°	14°	12°	12°	13°	12°
10°	280	13°	13°	13°	15°	14°	13°	14°	14°
15°	280	13°	14°	14°	12°	13°	13°	12°	15°
20°	280	14°	15°	14°	15°	14°	14°	12°	14°



Speed of Inclination
Fig. 2.—Goniogram (Normal).

used, the inclination with open eyes gives a greater average fall angle than that with closed eyes and the difference observed is 1° or less than 1° at each speed. Figure 2 shows the mean values from Tables I and II plotted in a graph. By plotting mean values from Tables I and II in a system of co-ordinates we obtained graphical representations, so-called goniograms (Fig. 2).

COMMENT

After Stein's method of goniometry had become known, the above mentioned investigators reported results obtained with hand-machine equipment such as in Watusji's and Kubo's methods. Their results generally have a common fault in that there is a wide range of individual variations. For instance, Kubo and Takasaki examined the fall angle in 70 normal persons and obtained an individual variation in a range of 33° max. 27° min. Such surprisingly wide variation may be caused by a defect in the equipment employed.

The figure of the variations obtained by Kubo's hand driven equipment is twice as large (33°) as that by our electrically driven equipment (15°).

The electrically driven apparatus has great advantage over the vestibular function test in that the individual variations are slight and consequently the difference between normal and pathological data is clearly demonstrated.

When the goniometrically normal persons are stimulated by electrically driven equipment (authors' method), the fall angles are directly proportional to the inclination speed and the results obtained are presented graphically. This graph is called a goniogram. Figure 2 shows the normal goniogram and in Part 2 of this article pathological goniograms will be reviewed.

Observations of the goniograms may make it easy to distinguish between normal and pathological vestibular (or equilibrial) functions.

On the other hand the rapid tilt test was first introduced as a clinical test by McNally and Tait[4] in 1926. Following a loss of one or both labyrinths, if the patient is placed on a tilt-table, it is very easy to upset him by a quick tilt to the side of the absent labyrinth. If the lesion is bilateral a tilt in any direction will upset him. McNally and Tait concluded that the absence of protective reaction to a quick tilt is due to a lesion of the semicircular canals. Moreover they[5] have given a full description of the parts of the labyrinth concerned in the response to a slow tilt, on the one hand, and to a quick tilt on the other.

Since McNally and Tait's work was published, Rademarker and Garcin[6] in 1932 and de Kleijn and Versteegh[7] in 1935 have used the quick-tilt test in the laboratory and the clinic as a means of diagnosing a loss of one or both labyrinths.

The rapid-tilt test appears to be an excellent method for demonstrating lesions of the individual vertical canals.

SUMMARY

With the electric goniometer devised by Honjo, 280 normal adults were examined for the angle of fall.

Subjects were inclined in four directions (anterior, posterior, right and left) at six different speeds of inclination (from 1° per sec. to 20° per sec.) with eyes both open and closed.

1. The averages, the maximum and minimum of the angle of fall in every direction, were in proportion to the speed of inclination and these can be represented as linear functions.

2. The difference between open and closed eyes at the same speed was less than the angle of 1° per sec.

3. The results obtained can be represented graphically. This graph is called a goniogram.

4. Individual variations of the angle of fall at the same stimulation is from 15° to 10°.

DATA OF 109 CASES OF IMPAIRED VESTIBULAR FUNCTION

Of the several controversial points regarding the results of goniometer test, the qualitative and quantitative differences between normal and pathological data are certainly not the least significant.

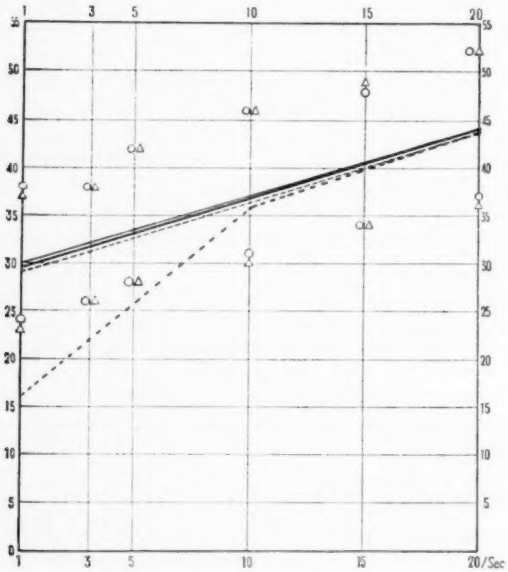
In an attempt to find an answer to this question, a careful study was made of 109 cases of vestibular dysfunction. These cases were chosen from a total series of 256 cases tested between May 1950 and April 1955 at Yamaguchi University Medical School. The cases were chosen otoneurologically on the basis of completeness of the records, anamnesis, and no cases were included in which repeated examinations were not made.

The number of selected patients was 109, of which 72 were males and 37 females, and the age ranged from 14 to 63 years, but the third and fourth decades of life accounted for the majority.

All these patients had vestibular dysfunction which was proved by such examinations as spontaneous nystagmus and rotatory, caloric and walking test, etc., and the following distribution of cases was made: Deaf mutism 51 cases, Ménière's disease 23 cases, labyrinthic trauma 18 cases, neuritis acustica 8 cases, acustic trauma 3 cases, otoxity of streptomycin 3 cases, labyrinthine syphilis 2 cases and acusticus tumor 1 case.

The goniometer tests were made by the author's apparatus, and the results obtained are shown by the graphical representations called goniograms (Fig. 2).

The results were classified into three general types according to the pattern of the goniogram, based upon the curves obtained with

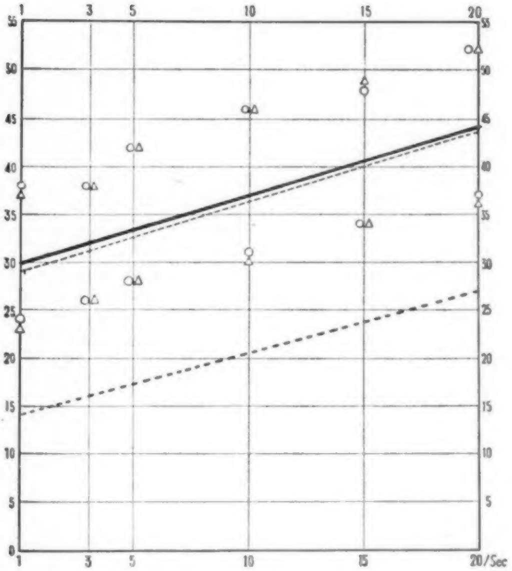


Open eyes —— Closed eyes - - - -
Fig. 3.—Type I of goniogram (Slight impairment case).

open eyes and closed eyes. If the curve with open eyes was within normal range, and the curve with closed eyes dropped from the normal range only at the low speeds of inclination such as 1°, 3° and 5° per sec., it was classified as Type I (Fig. 3) or slightly impaired. If the curve obtained with closed eyes dropped from the normal range at all the six inclination speeds tested, and the curve obtained with open eyes was exactly in the normal range, Type II, or moderate impairment was the designation (Fig. 4), and if there was a marked drop of both curves alike at the inclination speeds, Type III, or severe impairment was the designation (Fig. 5). The strict specifications for these classifications will be laid down in future by Furukawa.

Table IV shows the distribution of the three classifications among the total cases tested.

Type I containing the largest number of patients (66.9 per cent) showed a slight impairment which was often overlooked by other vestibular functional tests. This impairment was not recognizable



Open eyes —— Closed eyes - - - -
Fig. 4.—Type II of goniogram (Moderate impairment case).

until the patients with closed eyes were tested at low speeds of inclination such as 1°, 3° and 5°/sec. Type II, constituting 29.4 per cent, included those patients in whom vestibular dysfunction appeared only when they closed the eyes. Type III in which there was severe disturbance of vestibular function was represented by only a very small number of patients, 3.68 per cent.

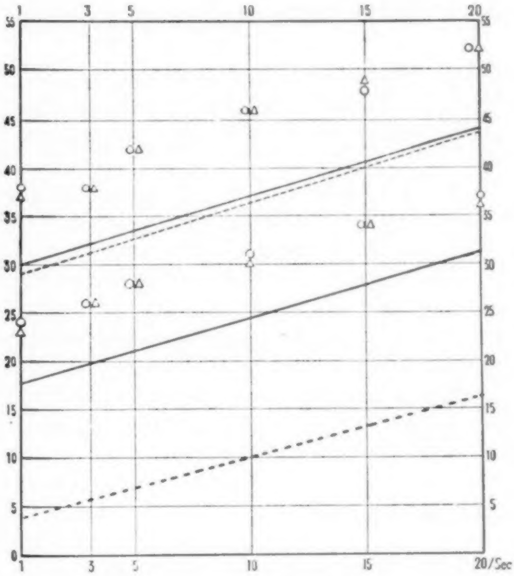
With regard to the distribution of diseases to types, it may be of some importance to observe that 50.9 per cent of the 51 deaf-mutes belonged to Type I, with slight vestibular impairment, and the remaining 49.1 per cent to moderate dysfunction, Type II. By contrast, 73.9 per cent of 23 subjects with Ménière's disease, 77.6 per cent of 18 labyrinthine trauma, and 75.0 per cent of eight neuritis acustica belonged to Type I, that is, in those three diseases Type I included about three-fourths of the total.

The small remaining group (i.e., ototoxicity of streptomycin 3, syphilitic labyrinthitis 2, acoustic trauma 3 and acusticus tumor 1) may be regarded as insignificant in the statistical series.

TABLE IV

DISTRIBUTION OF THREE TYPES OF GONIOGRAM AMONG THE TOTAL CASES

DIAGNOSIS		TYPE I		TYPE II		TYPE III	
	NUMBER	NUMBER	PER CENT	NUMBER	PER CENT	NUMBER	PER CENT
Deaf-Mutism	51	26	50.9	25	49.1		
Meniere's disease	23	17	73.9	4	17.4	2	8.7
Labyrinth trauma	18	14	77.6	3	16.8	1	5.5
Ototoxity of streptomycin	3	3	100				
Acusticus trauma	3	3	100				
Labyrinth syphilis	2	2	100				
Acusticus tumor	1			1	100		
Total	109	73 cases	66.9 per cent	32 cases	29.4 per cent	4 cases	3.68 per cent



Open eyes —— Closed eyes - - - -
Fig. 5.—Type III of goniogram (Severe impairment case).

The cases in each of three types were further studied to throw some light on the degree and quality of the vestibular impairment. For the purpose of comparing the results obtained by the goniometer with those of other vestibular functional tests carried out simultaneously, some cases belonging to each type were reviewed in greater detail.

REPORT OF CASES

Type I. Case 1. K. N., a male, aged 15. Diagnosis: Acquired deaf-mutism. There was no spontaneous or positional nystagmus. Rotation test (10 times in 20 seconds): rotation to the right, duration 8 seconds, frequency 9; rotation to the left, duration 9 seconds, frequency 9. Caloric test (10°, 20 cc): right ear, latent period 30 seconds, duration 70 seconds, frequency 21; left ear, latent period 25 seconds, duration 45 seconds, frequency 24. The walking test, Walting test and past-pointing were normal.

Results of goniometer test showed that the open eyes curve was normal and the closed eyes curve dropped only at the low speed (1°, 3°, and 5°/sec.) of inclination (Fig. 3, goniogram Type I).

Although the postrotatory nystagmus in this case presented a slight hypofunction in both labyrinths, results obtained by other vestibular tests showed no difference between the two labyrinths.

Case 2. M. Y., a male, aged 34. Diagnosis: Ménière's disease. He had been suffering from intermittent attacks of vertigo, vomiting and nausea with decrease of hearing and tinnitus in the left ear for the past years. His audiogram showed a moderately uniform hearing loss on the left side averaging 40-50 db, with normal hearing on the right side. There were no spontaneous or positional nystagmus. Rotation test (10 times in 20 seconds): rotation to the right, duration 19 seconds, frequency 41; rotation to the left, duration 21 seconds, frequency 40. Caloric test (10°, 20 cc): right ear, latent period 18 seconds, duration 107 seconds, frequency 213; left ear, latent period 29 seconds, duration 56 seconds, frequency 61. The walking test and Walting test were normal.

His goniogram showed practically normal curves both with open and closed eyes, but the dropping of the curve below the normal range (Fig. 3, goniogram Type I) was observed when the patient with closed eyes was inclined to the left or diseased side at a lower speed (1°, 3° and 5°/sec.).

Type II. Although the open eye curve of the goniogram in this type was normal, the closed eye curve dropped below the range at every speed inclination (Fig. 4, Type II).

Case 3. H. I., a male, aged 48. Diagnosis: Ménière's disease. The patient complained of intermittent buzzing and diminished hearing in the right ear for four months, and attacks of vertigo for three months. The latter recurred with increasing intensity. Medical treatment did not influence his vertigo and the patient was unable to work. Hearing tests were normal in the left ear, and markedly reduced and fluctuating in the left ear. There was spontaneous nystagmus (second degree). Rotatory and caloric nystagmus could not be observed owing to spontaneous nystagmus appearing at the same time.

The goniogram showed that the open eyes curve was within normal limits, while the closed eyes curve dropped below the normal

range at each speed of inclination (Fig. 4, goniogram Type II). It is noteworthy that inclining to the right or diseased side dropped, particularly, the curve of the fall angle.

CASE 4. F. I., a male, aged 47. Diagnosis: Labyrinthine trauma. The patient was admitted to a colliery hospital with symptoms of brain concussion following an injury received in a coal-truck accident, and was examined by us about five months following the accident. The patient complained of progressive deafness and continuous ringing in both ears, attacks of headache for the past month which had lately become very frequent and intense.

The audiogram showed a moderate hearing loss in both ears (averaging 70-80 db). There was spontaneous nystagmus (first degree). There was no response to the rotation test (10 times in 20 seconds) or the caloric test (10°, 20 cc). The goniogram showed the closed eyes curve at every speed, fallen below the normal range (Fig. 4, goniogram Type II).

Type III. CASE 5. I. F., a male, aged 44. Ménière's disease. The patient had been suffering from incapacitating attacks of vertigo for three years with a slight decrease in hearing and tinnitus in the left ear. The attacks recurred with increasing intensity and the gait was unsteady. The audiogram showed a slight hearing loss in both ears averaging 20-30 db. There was spontaneous nystagmus to the right side (first degree).

Rotation test (10 times in 20 seconds): rotation to the right, duration 37 seconds, frequency 86, with nausea; rotation to the left, duration 35 seconds, frequency 92, with nausea. Caloric test (17°, 20 cc): right ear, latent period 11 seconds, duration 169 seconds, frequency 316, with nausea; left ear, latent period 13 seconds, duration 184 seconds, frequency 331 with vomiting. The walking test and Walting test with closed eyes presented the so-called ataxic gait.

The goniogram revealed that both open and closed eyes curves presented a drop below normal limits (Fig. 5).

CASE 6. S. U., a male, aged 56. Diagnosis: labyrinthine trauma. The patient had been suffering from incapacitating attacks of vertigo for three months following an injury to the temporal bone sustained in an automobile accident. There was unsteady gait. The audiogram showed a more uniform hearing loss in the right ear, averaging 50-60 db with complete deafness on the left side. There was spontaneous

TABLE V

RELATION OF GONIOGRAM TYPES TO OTHER VESTIBULAR REACTIONS

	GONIOGRAM TYPE I	GONIOGRAM TYPE II	GONIOGRAM TYPE III
Subjective symptoms	Vestibular symptoms slight	Moderate	Severe
Spontaneous nystagmus	Usually negative	Present or negative	As a rule present or lost
Rotatory reaction	Hyporeaction or normally	Usually lost	Hyperreaction or lost
Caloric reaction	Hyporeaction or normally	Usually lost	Hyperreaction or lost
Walking, waltzing	Usually normal	Usually normal	

nystagmus (first degree). Rotation test (10 times in 20 seconds) and caloric test (10°, 20 cc): there was no response to either test in spite of repeated examinations. The walking test also presented the same result.

The goniogram showed the open eye curve at every speed dropped down from normal range while examination with closed eyes was impossible owing to ataxia.

COMMENT

Comparison of the results of the goniometer test and of other vestibular tests is reviewed in Table V. The Type I showed agreement with cases of slightly impaired vestibular function while Type II corresponded with these of moderate vestibular dysfunction. The Type III contained cases of severe vestibular hypo- or hyperfunction.

Regarding the cases with different vestibular function between the two ears, when the patient with hypofunction was inclined towards the affected ear, the curve of the goniogram showed a pronounced drop from the normal, and in the patient with hyperfunction the goniogram showed a drop from the normal when inclined toward the opposite side.

If one makes a comparison between the goniograms obtained by 280 normal healthy persons (Fig. 2) and the goniograms of the patients with altered vestibular function (Figs. 3, 4, and 5), it will be obvious that the qualitative distinction between the normal and pathological vestibular functions is not difficult.

From the results of our present goniometer tests, it is apparent that the analysis of the goniogram makes it possible to classify the vestibular dysfunction into three types far more clearly than would have been expected from the previous reports in the literature.

SUMMARY

With the new goniometer equipment devised by S. Honjo, 109 adults of both sexes with impairment of vestibular function were examined for falling angle. The speeds of inclination were 1°, 3°, 5°, 10° and 20° per second; the directions of inclination were forward, backward, right and left, and the tests were made both with open and closed eyes.

Results obtained were illustrated graphically by a goniogram, and may be summarized as follows:

1. Goniograms of vestibular patients are divided into three types.

2. Type I showed the curve with open eyes to be of normal range and that with closed eyes dropped only at low speeds of inclination.

3. Type II showed the curve with closed eyes to drop at every speed and to be normal with open eyes.

4. Type III showed both curves with open and closed eyes dropped at every speed.

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REFERENCES

1. Ikeda, S.: Diagnostic Value of Goniometer Test. Acta Clinic. Otolaryng. Kyoto 4:200-213, 1910.

2. Takasaki, M., and Kubo, I.: Function Test of Vestibular Organ. Bull. Fukuoka Acad. of Med. 7:558-563, 1919.

3. Kumagai, I.: Goniometer and Caloric Tests. Acta Clinic. Otolaryng. Kyoto 38:850-869, 1942.

4. Tait, J.: Ablation Experiments on the Labyrinth of Frogs. Laryngoscope 36:713-728, 1926.

5. McNally, W. J.: Report Upon an Experimental Investigation of the Equilibrial Mechanism of the Frog's Labyrinth. Tr. Am. Acad. Ophth. 36:283-304, 1931.

6. Rademaker, G. J., and Garcin, R.: Note sur quelques rèactions labyrinthques des extrèmitès chez l'animal et chez l'homme. Rev. Neurol. 1:637-653, 1932.

7. de Klein, A., and Versteegh, C.: Ueber labyrinthaere Gleichgewichtsreaktionen bei Menschen und Tieren nach schnellen Kippen um die longitudinale Achse. Acta Oto-laryng. 24:34-52, 1935.

XXXIV

SALIVARY ADENOMAS OF THE BUCCAL CAVITY

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Over a period of years, clinical observation revealed that some of the salivary adenomas arising within the buccal cavity ran an unsatisfactory course and ultimately presented a grave prognosis. In some of the cases, late genuine regional and distant metastases occurred where the primary tumor had been classed among the nonmalignant mixed adenomas. It was following these observations that interest and curiosity developed about this group of neoplasms.

A large series of sixty-three salivary tumors of the buccal cavity have been investigated and treated at the Christie Cancer Hospital and Holt Radium Institute, Manchester, England, and the Department of Otolaryngology, Manchester Royal Infirmary, and follow-up records have been available in all the cases. The tumors have been classified into the following types according to the histological pattern:

1. Mixed adenomas (27 cases).

2. Adenomas showing cylindroma pattern (36 cases).

This paper is mainly concerned with the cylindromas which are undoubtedly malignant tumors and a most formidably lethal group. Their behavior is quite unpredictable when compared with the mixed adenomas. They run a slowly progressive course and their mode of spread is similar to that of a rodent tumor. Unless adequately removed, relentless recurrence takes place. Survival may last for many years with eventual severe morbidity. Metastases may occur at a late stage, both in regional nodes and distant sites. The cylindroma may masquerade as a mixed adenoma, so it is very important to differentiate between them, and much wiser to confirm the diagnosis histologically before treatment is carried out.

Read at the Sixth International Congress of Otolaryngology, Washington, D. C., May 1957.

The buccal cavity is a common ground for many different specialists and, as a result, these tumors are handled by general surgeons, radiologists, dental surgeons and laryngologists, and because of their rarity, one clinician may see very few cases in a lifetime's work.

The cylindroma has received many different synonyms, e.g., adenoid cystic carcinoma (Spies), basalioma (Krompecher), adeno-carcinoma of mixed tumor type (New), and it has also been likened to skin tumors of the epithelioma adenoides cysticum pattern. This confusion of terminology has been misleading, and has resulted in a lack of understanding of the dangerous nature of this tumor.

HISTORICAL SURVEY

Billroth's paper in 1859 is a distinct landmark. He first used the term "cylindroma" to describe a tumor of the parotid region which recurred many times during a period of twenty years: it was operated on nine times and finally remained untreatable. Malassez (1883) integrated the subject of cylindromas by enumerating the eleven synonyms that had been used to describe them. Stephen Paget (1886) was one of the first to describe "adenomata" arising on the palate. Ribbert (1907) gave an authoritative ruling on the histological structure of cylindromas and also on their clinical behavior. He believed the cylindroma to be an epithelial tumor arising in the mucous gland ducts, and described one case where the tumor had destroyed the hard palate, slowly invaded the base of the skull, and finally metastasized to the lungs. Krompecher (1908) classed cylindromas with basal cell tumors of the skin, and called them all "basal cell cancers" and first used the term "basalioma." Spies (1930) reviewed a large series of cases of adenoid cystic carcinoma which he thought resembled tumors of the epithelioma adenoides cysticum pattern. The illustration of one of these is undoubtedly a cylindroma. He thought the prognosis was very bad and three of his cases developed generalized metastases. New and Childrey (1931) reported a large series of mixed tumors of the tonsil and pharynx, and called them adeno-carcinomas of mixed tumor type. Three of their cases developed late cervical metastases. Ahlbom (1935) in his monograph on "Mucous and Salivary Gland Tumors" called the tumors of cylindroma pattern "basaliomas." Lemaitre (1936) used the term "cylindroma" and pointed out the importance of adequate removal the first time and stressed the grave prognosis. Ringertz (1938) adopted Krompecher's term "basalioma" for what is, in effect, the cylindroma pattern, in his treatise on "Malignant Tumors in the Nasal and Paranasal Cavi-

ties." Kramer and Som (1939), reporting on cylindromas of the upper air passages, considered the tumor was related to basal cell carcinoma, and thought it to be locally malignant and rarely producing distant metastases. Docherty and Mayo (1942 and 1943) appreciated that cylindromas were confused frequently with mixed tumors. They described the malignant tendencies of the cylindroma and emphasized that metastases occurred. Mulligan (1943) describing metastases of mixed tumors of the salivary glands, realized the confusion that had arisen because of the different synonyms used. He accepted the term "cylindroma" and in some of his cases there were widespread metastases. Pollack (1952) described five cases of cylindroma in the nose and sinuses and stressed the persistent and relentless nature of their recurrence and the possibility of widespread metastases. He, too, thought that radical removal should be the initial form of treatment. Lambert (1953) thought that salivary adenomas outside the large salivary glands arose most commonly on the hard and soft palate, and there was a definite preponderance of the disease in the female. He, too, stressed wide excision. Foote and Frazell (1953), in their study of over five hundred tumors of the major salivary glands, described the cylindromas as "adenoid cystic carcinomas." The illustrations certainly showed the cylindroma pattern. They noted the multiple recurrence of the tumor with late generalized metastases. Berdal and Mylius (1954) used the term cylindroma in describing twelve cases occurring in the upper respiratory and digestive tracts. They considered the tumors to be radioresistant and advised radical surgical treatment. Russell (1955) described the pathological aspect of a large collection (256 cases) of adenomatous tumors of the anterior foregut region. She thought the name "cylindroma" should be accepted, for the tumor is now correlated with a special clinical picture and prognosis. Harrison (1956) reported a large series of "ectopic" salivary adenomas, following a long term clinico-pathological study. He adopted the term "cylindroma" and stressed the malignant nature of the tumor and the grave ultimate prognosis. He advised radical excision when the tumor was surgically accessible. Ryan (1957) advised radiological investigation to help disclose any extensions of a cylindroma not evident clinically, and also a chest x-ray to reveal any unsuspected metastases.

HISTOLOGY

The mixed adenoma showed a pattern similar to the ones arising in the parotid gland, that is, of a typical mixed histology of epithelial and mesenchymal elements.

Fig. 1.—High power view of cylindroma to show detail of the cylinder-like spaces. 110 x magnification. This section was specially prepared by the Periodic-acid Schiff stain in order to define the cylinder spaces. The cylindrical spaces can be clearly seen and from within outwards there is the central blob of mucus, and successively an inner hyaline layer, then an epithelial cellular ring, an outer hyaline layer and finally connective tissue septa. The hyaline layers are clearly visible like haloes.

The cylindroma has a characteristic pattern which is quite distinct and unique. It is an epithelial tumor with adenoid structure, and appears to arise in the mucous gland ducts which are abundant in the mucous membrane lining the buccal and nasal cavities. The tumor is rarely encapsulated, and it infiltrates the surrounding tissues. Figure 1 shows in detail the cylinder spaces which were the source of the descriptive term "cylindroma."

SITE INCIDENCE

Ectopic salivary adenomas occur most commonly within the buccal cavity. Of the total sixty-three cases, two-thirds arose on the hard and soft palates, but the former is by far the commonest site. This finding is in agreement with previous publications. Six of the cases were recurrent when first seen and they had recurred at any time up to eight years after treatment.

Fig. 2.—Mixed adenoma. Swelling right soft palate which recurred ten years after treatment by deep x-ray therapy.

The tumors in the tongue and floor of mouth were all of the cylindroma type.

SEX AND AGE INCIDENCE

In the mixed adenoma group the sex incidence was about equal, while the cylindromas were more common in females. They all occurred in adults, and were most common in the fifth and sixth decades.

CLINICAL FINDINGS AND COURSE OF THE MIXED ADENOMAS

The only symptom was a painless swelling, often only noticed by chance (Figs. 2 and 3). The swelling was usually elastic in consistency and sometimes cystic. Ulceration of the mucosal surface was only found in one case.

The mixed adenomas behaved as non-malignant tumors and their progress was very slow indeed.

Enucleation of the tumor was the treatment of choice. Some of these tumors were shaped like an artichoke, and it was important to remember this when carrying out the extra-capsular dissection. When-

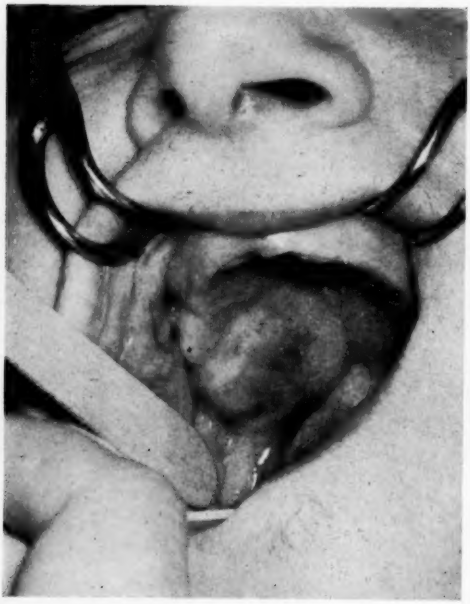

Fig. 3.—Mixed adenoma. Swelling posterior part right hard palate extending backwards; noticed accidentally.

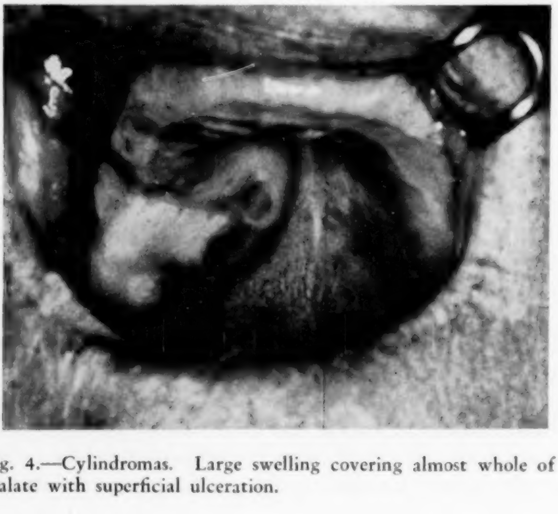

Fig. 4.—Cylindromas. Large swelling covering almost whole of right hard palate with superficial ulceration.

Fig. 5.—Swelling left soft palate with marked dilatation of the superficial vessels.

ever there was any doubt about complete removal, post-operative irradiation was used, and this happened in only four of the cases. The choice of irradiation depended on the site, and implantation of gold seeds or radium needles or deep x-ray therapy was used.

The prognosis is very good indeed. Of the twenty-seven patients, twenty-one are alive and well anything from four to sixteen years after treatment. Three patietns have died from intercurrent disease and three have not been traced.

CLINICAL FINDINGS AND COURSE OF CYLINDROMAS

In the palatal cases, there was a swelling, frequently associated with pain. Ulceration was quite a common feature due to invasion of the surface epithelium by tumor (Fig. 4). Often the surface of the tumor showed a marked dilatation of the superficial vessels (Fig. 5).

These clinical features may be most helpful to differentiate the dangerous cylindroma from the simple mixed adenoma. The tumor is quite variable in size and firm in consistency, but this does not help in differential diagnosis.

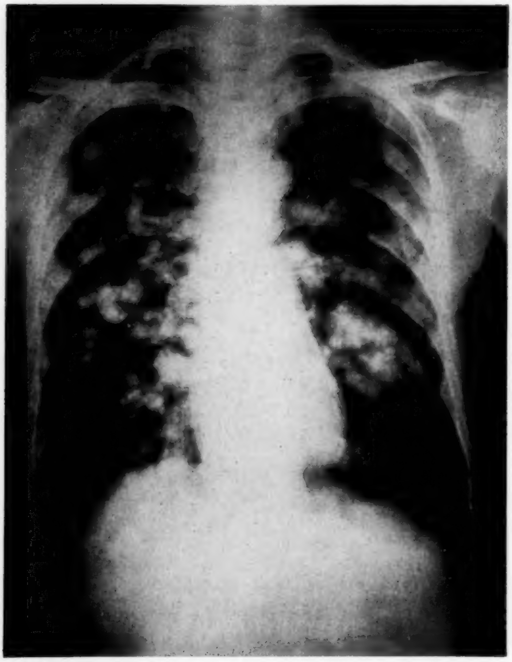

Fig. 6.—Latent metastases in the lungs from a recurrent cylindroma of the palate.

The clinical course of the cylindroma is quite characteristic. The tumor grows slowly, commonly ulcerates and is locally invasive and infiltrating. Unless the primary tumor is widely excised when accessible, there is relentless local recurrence and may be late invasion of the middle fossa of the skull. When this occurs, the gasserian ganglion and its branches may be involved and this results in neuralgic pain in different parts of the face and head, resulting in prolonged and severe morbidity. Metastases of the regional lymph nodes and distant spread to the viscera and skeleton may occur.

In this series there was cervical lymph node involvement in six cases, and the glands were observed at varying periods up to twelve years after treatment of the primary. There was also distant spread in six cases, three of them metastasizing to the lungs (Fig. 6), one of which also had deposits in the ilium. The lung metastases may be

TABLE I

MIXED ADENOMAS	NO. OF CASES	CYLINDROMAS	NO. OF CASES
Hard Palate	13	Hard Palate	17
Soft Palate	10	Soft Palate	4
Lips	3	Lips	1
Buccal Mucosa	1	Buccal Mucosa	2
		Floor of Mouth	4
		Tongue	
		(a) dorsum	2
		(b) base	6
	27		36

quite latent and unsuspected, and a chest x-ray should always be done. Radiological observation has shown that these metastases grow very slowly, and some patients have survived with them over a period of years. In the other three cases there was spread to the liver in one and to the ilium and a cerebral hemisphere in the others. These distant metastases occurred at varying times up to five years after treatment of the primary tumor.

The course of the cylindroma is undoubtedly much slower than that of the frank carcinoma occurring in similar sites.

In this series of thirty-six cases, ten of them were recurrent when first seen. In each case excision had been performed, and the tumors had recurred any time up to fourteen years afterwards. It is interesting to note that these recurrences were in surgically accessible sites. It is my belief that when treated the dangerous characteristics of the tumor were not understood, and so the excision had not been sufficiently radical.

TREATMENT

Over the years, various forms of treatment were used including different forms of irradiation, surgical methods and sometimes a combination of both.

On review, the surgical method is the one of choice, and when the tumor is accessible, as wide an excision as possible should be carried out. It is important to stress wide excision, as frequently there is no true capsule, and the tumor has spread outside naked eye limits. This has been noted in some cases on histological examination.

With a tumor arising on the hard palate, the overlying palatal bone requires removal, otherwise the excision will be inadequate and it will certainly recur. This has happened in our experience with disastrous results. At the end of the operation a dental obturator (Fig. 7) is applied, so that the patient can eat and talk quite satisfactorily with only limited disability immediately on recovering from the anesthetic. Later, the obturator is replaced by a permanent prosthesis which the patient can wear like any other dental plate. These prostheses can also be made for operative defects of the soft palate (Fig. 8).

PROGNOSIS

This is certainly most grave.

Of the thirty-six patients, two-thirds (twenty-four) have died with recurrence, all except one within seven years of treatment of the primary.

Four patients are alive with an inoperable recurrence.

There are only three patients alive and well, one for ten years (floor of mouth), another for nine years (base of tongue) and the third for four years (buccal mucosa).

Three of the patients died from intercurrent disease and two have not been traced.

SUMMARY

A survey of seventeen years' collection of salivary adenomas of the buccal cavity (63 cases) showed that the cylindroma was more common than the mixed adenoma and had a graver prognosis. Two-thirds of the tumors arose on the hard and soft palates and the former was undoubtedly the commonest site.

Clinically, the cylindroma often masquerades as a mixed adenoma. Ulceration and pain occur frequently in the cylindromas and the surface of the tumor may show very marked vascularity, thus, careful examination may be most helpful in the differential diagnosis. It is wiser to identify the cylindroma from a representative biopsy before carrying out treatment, and the surgical pathologist must be familiar with its characteristic pattern and fully alive to its significance.

The cylindromas are malignant tumors and when surgically accessible, radical excision is indicated. Unless the tumor is adequately

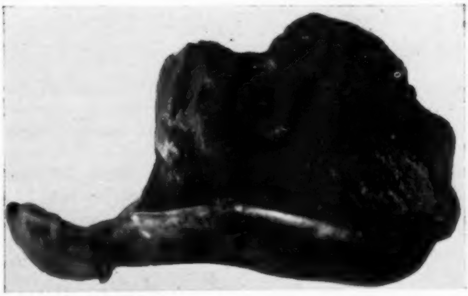

Fig. 7.—This type of dental obturator is a temporary prosthesis applied after removal of the palatal bone for a cylindroma. It consists of a clear acrylic base plate made from a pre-operative impression. The obturator portion is modelled from black gutta percha so that it fits satisfactorily into the operative defect and is then attached to the base plate.

Fig. 8.—The type of permanent prosthesis which is used for an operative defect of the soft palate.

TABLE II

CYLINDROMAS—SURVIVAL RATE

SITE	YEARS OF SURVIVAL	NO. OF CASES	END RESULTS
Hard Palate (17 cases)	19	1	Alive with recurrence
	15	1	Died with recurrence
	12	2	1 alive with recurrence and cervical nodes 1 alive with recurrence
	10	2	1 died—intercurrent disease 1 alive with recurrence
	7	1	Died with recurrence
	6	1	Died with recurrence
	5	1	Died with recurrence
	under 5	8	5 died with recurrence 1 died with recurrence and metastases in the lungs, ilium and cervical nodes 1 died with recurrence and cervical nodes 1 died with recurrence and metastases in the lungs
Soft Palate (4 cases)	8	1	Alive with recurrence
	under 5	3	1 died with recurrence 1 died with recurrence and cervical nodes 1 not traced
Tongue-Base (6 cases)	9	1	Alive and well
	7	1	Died—intercurrent disease
	5	1	Died with recurrence and metastases in the ilium
	under 5	3	1 died with recurrence and brain metastases 1 died with recurrence and cervical nodes 1 died—intercurrent disease
Tongue-Dorsum (2 cases)	under 5	1	Died with recurrence and metastases in the lungs and cervical nodes 1 not traced
Floor of Mouth (4 cases)	10	1	Alive and well
	5	2	1 died with recurrence 1 died—intercurrent disease
	under 5	1	1 died with recurrence and liver metastases
Buccal Mucosa (2 cases)	under 5	1	Alive and well Died with recurrence
Upper Lip (1 case)	under 5	..	Died with recurrence

removed, relentless local recurrence occurs. Infiltration and invasion takes place and the mode of spread is similar to that of a rodent tumor and there is severe and prolonged morbidity. Some tumors have recurred as long as fourteen years after treatment. Because of late recurrence or development of metastases, prolonged follow-up is essential.

In the present series, the cylindroma has been mostly resistant to irradiation, but in spite of this, when the tumor is surgically inaccessible, it is worth while trying one of the newer forms of irradiation, as an odd case responded temporarily.

There is no real difficulty in the management of the mixed adenomas, except in the rarely inaccessible ones. Enucleation is the treatment of choice, and post-operative irradiation is only used whenever there is any fear of residual tumor.

Finally, Billroth's term "cylindroma" has such high priority historically and is so descriptive of a characteristic adenoma pattern that, if generally used by clinicians and pathologists, the confusion in terminology could be avoided, and this would result in a better understanding of what has always been a very difficult problem.

Clinical Sciences Building

BIBLIOGRAPHY

Ahlbom, H. E.: Mucous and Salivary Gland Tumors. Acta Radiol. (Stockh.), Suppl. 23, 1935.

Berdal, P., and Mylius, E.: Cylindromas of the Respiratory Tract, the Upper Part of the Digestive Tract and Adjoining Organs. Acta Oto-Laryng. (Stockh.), Suppl. 118:32, 1954.

Billroth, T.: Beobachtungen über Geschwülste der Speicheldrüsen. Virchows Arch. Path. Anat. 17:357, 1859.

Docherty, M. B., and Mayo, C. W.: Primary Tumors of the Submaxillary Gland with Special Reference to Mixed Tumors. Surg. Gynec. Obstet. 74:1033, 1942. Cylindroma—Report of Two Casese with Metastasis. Surgery 13:416, 1943.

Foote, F. W., and Frazell, E. L.: Tumors of the Major Salivary Glands. Cancer 6:1065, 1953.

Harrison, K.: A Study of Ectopic Mixed Salivary Tumors. Ann. R. Coll. Surg. Engl. 18:99, 1956.

Kramer, R., and Som, M. L.: Cylindroma of the Upper Air Passages. Arch. Otolaryng. (Chicago) 29:356, 1939.

Krompecher, E.: Zur Histogenese und Morphologie der Mischgeschwülste der Haut Sowie der Speichel - und Schleimdrüsen. Beit. Path. Anat. 44:51 and 88, 1908.

Lambert, V. F.: Mixed Salivary Tumors of the Upper Respiratory Tract. J. Laryng. 67:603, 1953.

LeMaitre, F., et al.: Introduction a l'etude des Tumeurs Dites Cylindromes. Acta Oto-Laryng. (Stockh.) 24:112, 1936.

Malassez, L.: Sur le Cylindrome. Arch. Phys. Norm. Path. 3s. 1:186, 1883.

McDonald, J. R., and Havens, F. Z.: A Study of Malignant Tumors of Glandular Nature Found in the Nose, Throat and Mouth. Surg. Clin. N. Amer. 28:1087, 1948.

Mulligan, R. M.: Metastasis of Mixed Tumors of the Salivary Glands. Arch. Path. 35:357, 1943.

New, G. B., and Childrey, J. H.: Tumors of the Tonsil and Pharynx—Adeno-Carcinomas of the Mixed Type. Arch. Otolaryng. (Chicago) 14:669, 1931.

Paget, S.: Tumors of the Palate. St. Bart's Hosp. Rep. 21:315, 1886.

Pollack, R. S.: Cylindroma in Nose and Sinuses. Arch. Otolaryng. (Chicago) 55:210, 1952.

Ribbert, M. W. H.: Ueber das Zylindrom. Dtsch. Med. Wschr. 33:126, 1907.

Ringertz, N.: Pathology of Malignant Tumors Arising in the Nasal and Paranasal Cavities and Maxilla. Acta Oto-laryng. (Stockh.) Suppl. 27, 1938.

Russell, H.: Adenomatous Tumors of the Anterior Foregut Region Showing the Cylindroma Pattern. Brit. Jour. Surg. 43:248, 1955.

Ryan, M. J.: Radiological Manifestations of Ectopic Salivary Adenomas Showing the Cylindroma Pattern. Proc. R. Soc. Med. 50:96, 1957.

Spies, J. W.: Adenoid Cystic Carcinoma. Arch. Surg. (Chicago) 21:365, 1930.

XXXV

ANOMALIES IN THE RECORDED MOVEMENTS OF THE EYE DURING OPTOKINETIC ROTATORY AND CALORIC STIMULATION IN NORMAL SUBJECTS

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and

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The object of this paper is to demonstrate, as far as it is possible, the normal reactions, and their variations in certain apparently normal subjects, to optokinetic stimulation of the eye muscles, and to rotatory and caloric stimulation of the vestibule. The reaction consists of nystagmoid movement of the eyeballs, and is recorded by picking up, by means of electrodes applied to the skin at the outer corners of the two orbits, the variations in the field of the corneoretinal potential, caused by movements of the eyeballs. These variations are suitably amplified and are inscribed, on a moving strip of paper, by a pen recorder. This is so arranged that the amplitude of the eye movements and the speed of the slow phase can be readily determined.

The advantages of the electrical recording of the eye-movements over the visual method are that the beginning and end points of the movements, their amplitude and their speed can be more easily and more accurately estimated and can be permanently recorded on paper. The movement of the eyes can be recorded equally well with the eyes open or closed, during rotation in a revolving chair or when the subject is surrounded by a revolving drum.

THE OPTOKINETIC TEST

The diagnostic value of the optokinetic test, in which a black and white striped drum is rotated in front of or around the patient, has long been recognized, and its importance has recently been em-

Read at the Sixth International Congress of Otolaryngology, Washington, D. C., May 1957.

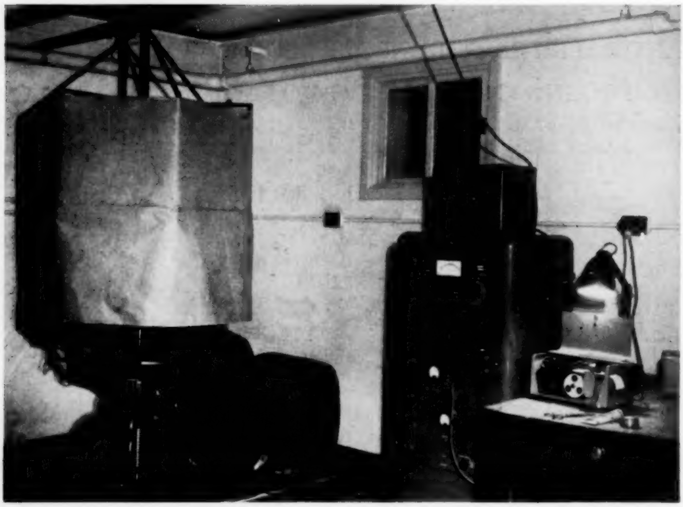

Fig. 1A.—View of apparatus used in vestibular testing, with the optokinetic drum in position. The rotating chair, control unit and pen recorder can also be seen.

phasized by Carmichael and Cawthorne.[1] Some preliminary investigation of the results of optokinetic tests, using the graphic recorder, showed that precise control of the speed of the drum did not yield regular nystagmoid strokes, but that there was a considerable random variation. A hollow hexagonal drum has been evolved which can be revolved around the subject at a velocity, with acceleration and deceleration all of which can be predetermined and controlled by electronic means (Fig. 1A). The inside of the drum is lined with matt paper of a neutral color and there are six vertical white stripes (Fig. 1B), which are of such width that the image produced by a single stripe exactly covers the macula. It is easy for a subject to fix the gaze onto these stripes as they traverse the visual field and follow them accurately. The subject can comfortably fix his gaze onto the succeeding stripes of the rotating drum for several minutes. The nystagmoid movements of the eye which arise from this stimulus are of a very regular form as shown in Figure 2. When the speed of the slow phase of the eye-movement is measured it is found to agree with the speed of the drum to within about 10 per cent, which can be explained by small experimental inaccuracies.

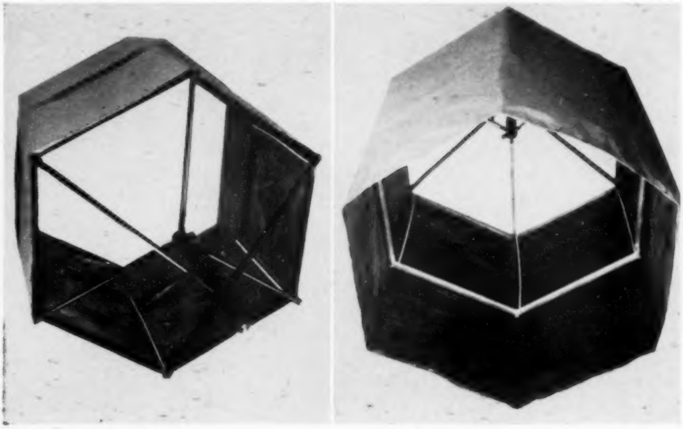

Fig. 1B.—Optokinetic drum showing the interior.

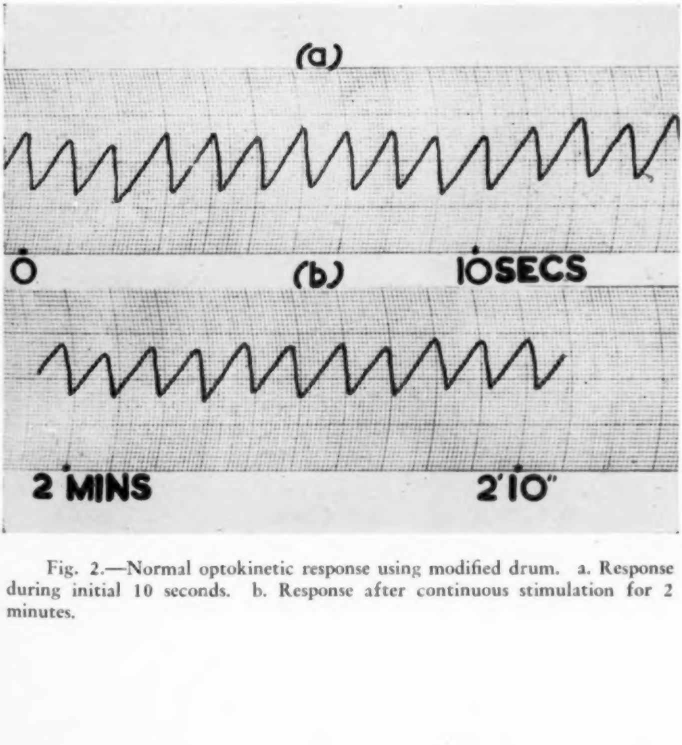

Fig. 2.—Normal optokinetic response using modified drum. a. Response during initial 10 seconds. b. Response after continuous stimulation for 2 minutes.

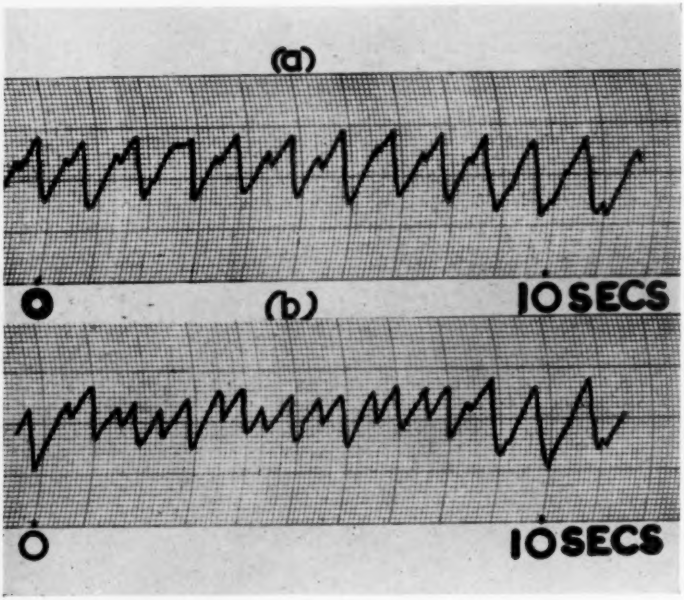

Fig. 3.—Optokinetic response with conventional drum, showing variations in the speed of the slow phase.

When the more conventional drum—with wider stripes—is used the graphic record shows variations in speed from one slow phase to another and even within the range of one slow phase. This is probably due to the gaze being less accurately fixed, moving from the leading to the trailing edge of the stripe and perhaps at random across its width. The result is shown in Figure 3 when the variation in speed between different strokes is seen and also the change of speed during a single slow phase.

It was thought that if an ideal response to the optokinetic variation could be obtained, it would be possible to establish a preponderance to either side. It has been found, however, that with the present form of drum, when the observer closes his eyes, there is a cessation of nystagmus for a short time followed by a resumption of nystagmoid movements in the same direction as the primary nystagmus (Figs. 4 and 5). This has been described as "after nystagmus" and may be comparable to perrotatory nystagmus to be described later. The

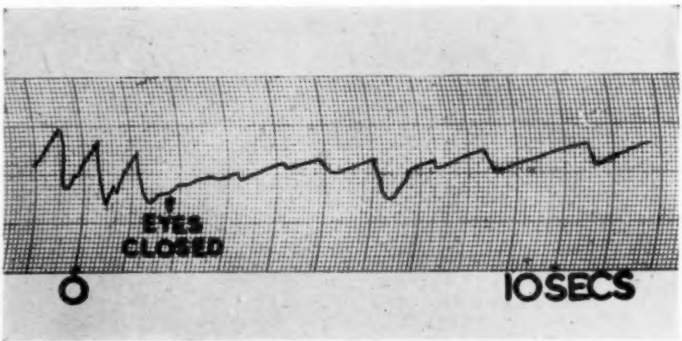

Fig. 4.—Normal after nystagmus upon closing eyes.

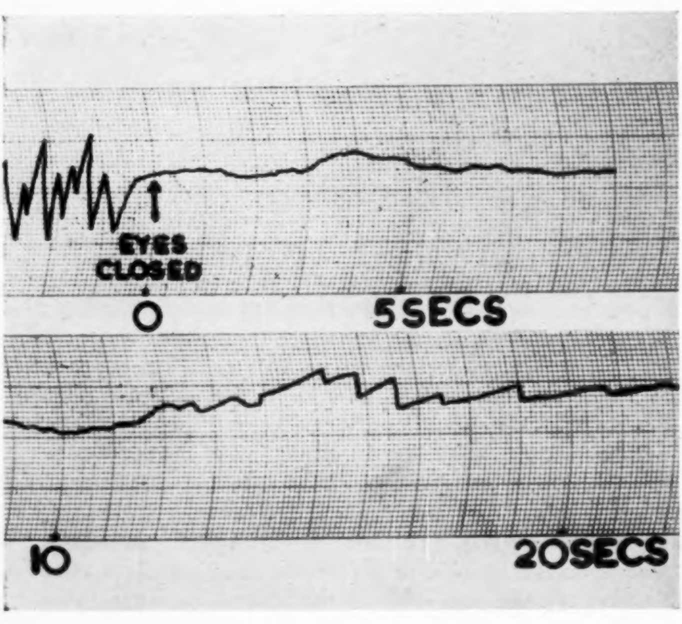

Fig. 5.—Showing delay in onset of normal after nystagmus.

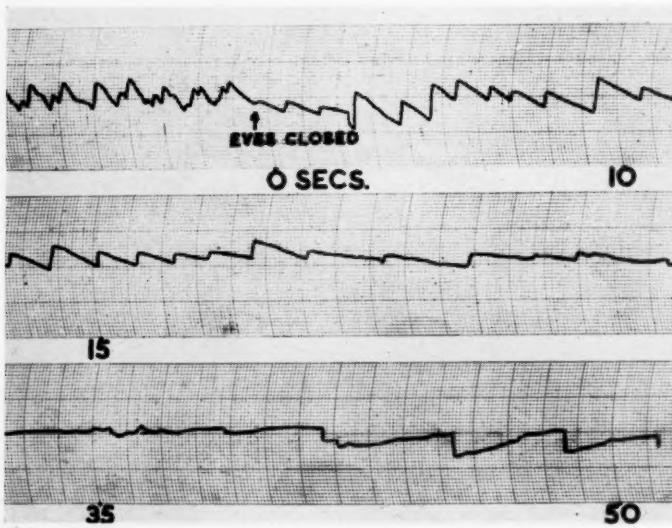

Fig. 6.—Shows reversal of normal after nystagmus.

The mechanical analysis of the semicircular canal system suggests that deviation of the cupula results in nystagmoid movements of the eye, the speed of the slow phase being proportional to the degree of cupular deviation. This is true of young healthy normals who have not experienced rotation, but it is not necessarily true of those who have been frequently rotated. Such are expert skaters, ballet dancers and scientists and their assistants who rotate themselves experimentally. After repeated rotation in normals and in patients suffering after nystagmus may persist for variable periods up to one minute and in a certain number of apparently normal subjects it is followed by a short quiet period and then a nystagmus in the opposite direction (Fig. 6) which has been described as nach nach (after after) nystagmus but which is comparable to the secondary nystagmus which is observed after rotation. The term "contra-nystagmus" may be used to denote this late nystagmus of reversed direction. It is a little difficult to say whether this effect is the normal one or is an anomaly. It may be that the effect is due to the stimulation of a latent spontaneous nystagmus. It is found that this "contra-nystagmus" is often obtained in individuals who show some directional preponderance to caloric testing—which may be considered to give some support to this

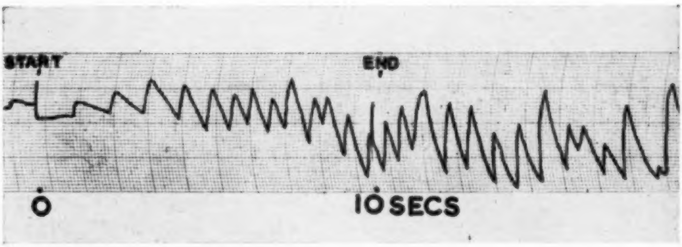

Fig. 7.—Normal response during acceleratory stimulus. "START" and "END" refer to the duration of the stimulus 10°/sec² for 10 seconds.

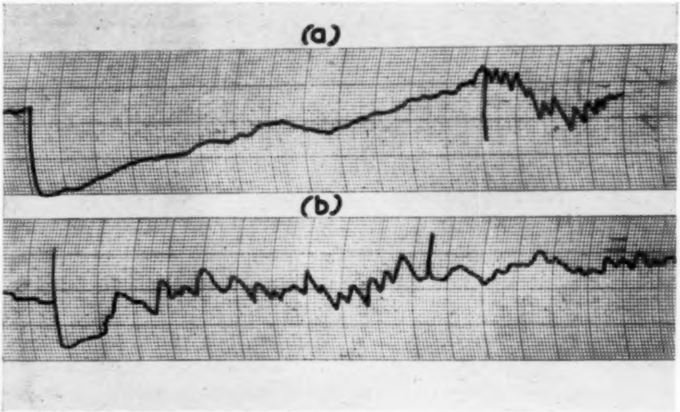

Fig. 8.—Anomalies of response during acceleration. a. Suppression of nystagmus during stimulation. b. Suppression of nystagmus following stimulus.

suggestion. In some normal subjects there may be a) no after nystagmus or b) there may be a delayed onset of after-nystagmus.

The contra-nystagmus may last for one minute or more and then undergoes a rapid decay.

ROTATIONAL NYSTAGMUS

from end organ disease with consequent spontaneous nystagmus the relation between slow phase velocity and the degree of cupular deflec-

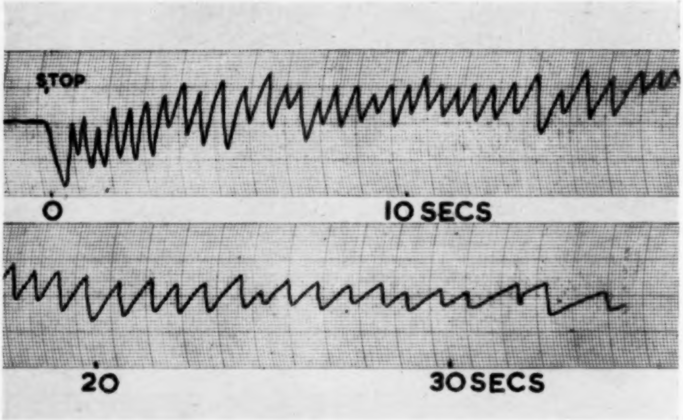

Fig. 9.—Normal nystagmus following a sudden stop from 100°/sec constant velocity.

tion is not maintained. In some cases, indeed, only a few nystagmoid jerks result from a sudden stop from 100 degrees per second. The normal response to an acceleration of 10 degrees per second per second for 10 seconds is shown in Figure 7 and may be described as the primary nystagmus, and the gradual build up of the slow phase from an almost imperceptible first stroke is demonstrated. The gradual decay after cessation of acceleration is also shown.

Certain anomalies which are not infrequent are shown in Figure 8. The first is an inhibition of nystagmus during acceleration, the nystagmus only beginning when a steady velocity has been reached. The second shows a suppression immediately on reaching the steady velocity followed by a resumption of nystagmus. Figure 9 shows the normal response to a sudden stop after a period of immobility of the eyes during rotation at a steady velocity. The immediate onset of nystagmus is shown, with a gradual slowing down of the speed of the slow phase. This reduction in the speed and in the amplitude of the stroke reflect the gradual movement back to its resting position of the cupula. In Figure 10 are shown two forms of anomalous response. The first shows only a few strokes of nystagmus on sudden stopping of rotation followed by complete suppression. The second shows an inhibition of response followed by a resumption of normal nystagmoid excursions. It is possible for various combinations and

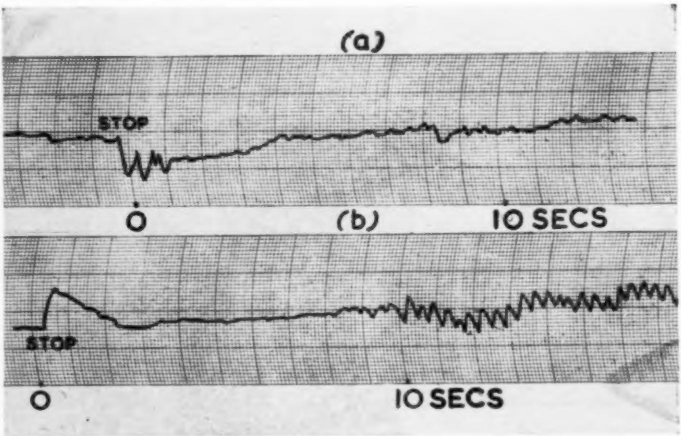

Fig. 10.—Anomalies of response after sudden stop. a. Suppression of nystagmus following two or three strokes. b. Return to normal nystagmus after suppression for the initial 10 seconds following the stimulus.

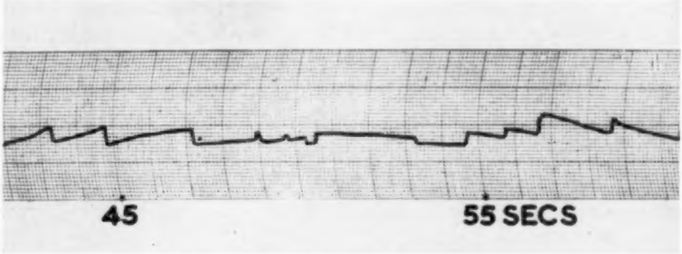

Fig. 11.—Outset of secondary nystagmus. The transition period is characterized by quick phase jerks in both directions, giving rise to square waves.

variations in these two anomalies to occur in normal subjects and suspicions of a pathological cause may readily be aroused if the existence of these anomalies is not appreciated.

Secondary Nystagmus. An effect which was at first regarded as an anomaly but may really be regarded as normal was the emergence of a secondary nystagmus in the reverse direction to the primary one. It has been shown that this is so constant during rotation tests that it can be accepted as part of the normal response (Fig. 11).

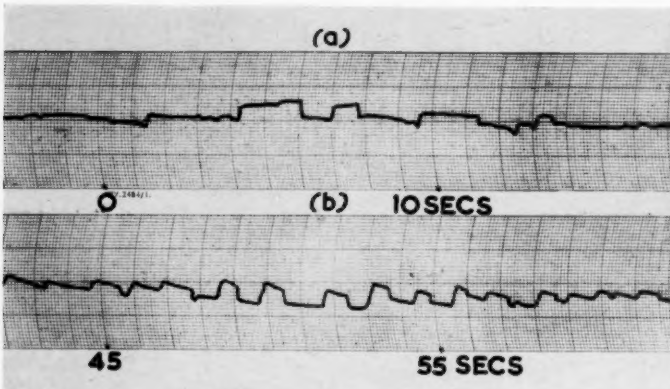

Fig. 12.—Examples of square waves obtained during positional tests in two different patients.

There may be a short quiet period between the primary and secondary nystagmus or there may be a period of "square waves" where the nystagmoid movements consist in alternating quick movements in either direction separated by pauses but accompanied by no slow movements. These square waves signal the onset of secondary nystagmus and could be explained by the co-existence of two nystagmoid movements in opposite directions. These square waves are also found in individuals suffering from a number of pathological conditions and Figure 12 shows a recording of square waves found during head positional tests in two such patients.

CALORIC NYSTAGMUS

The normal nystagmoid response to caloric stimulation of the semicircular canal system begins within five or ten seconds from the beginning of the douching. At first there is an almost imperceptible slow phase followed by a quick jerk and then a rapid build up to the normal rhythm. This has usually been reached before the end of the forty seconds stimulation and continues for about three minutes when a progressive decay in the amplitude and speed sets in and lasts for a further two minutes as recorded electrically (Fig. 13). This period is some two minutes longer than can usually be detected by visual observation of the eyeballs.

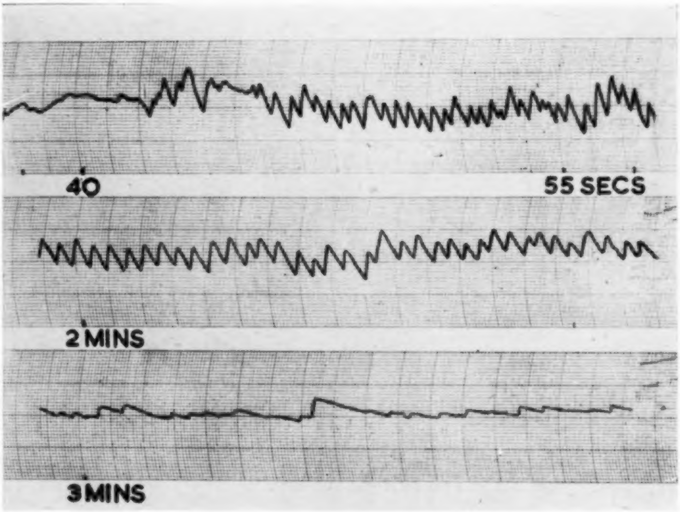

Fig. 13.—Normal response to caloric stimulation.

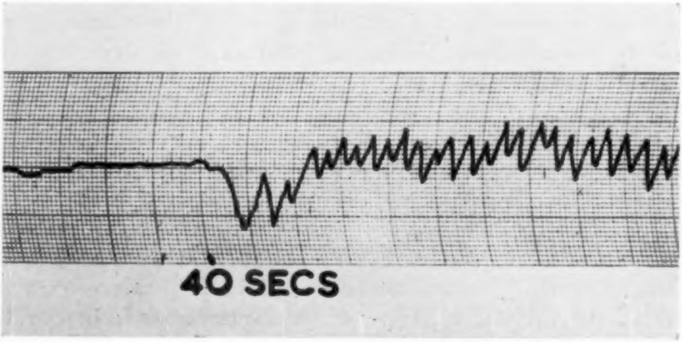

Fig. 14.—Suppression of nystagmus during douching.

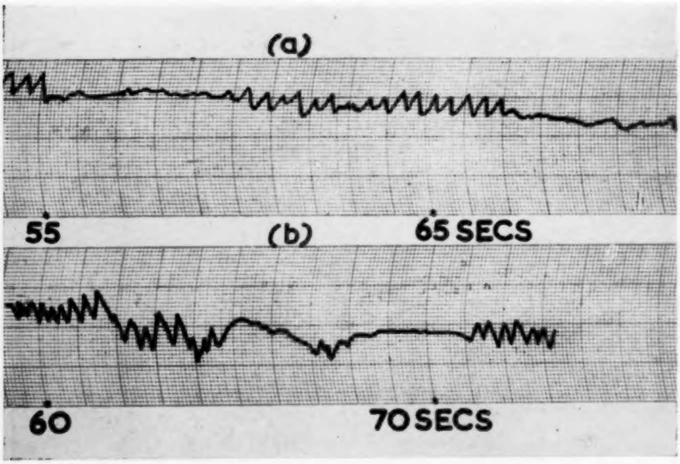

Fig. 15.—Anomalies of response during caloric test. Intermittent suppression of nystagmus in two different subjects.

In a number of apparently normal subjects there is a delay in the onset of nystagmus which may not begin until the end of the stimulatory douching (Fig. 14). This may be considered as a suppression or inhibition of nystagmus and may be a subjective response to the dislike of the tactile stimulus set up in the meatus by the douching. It is followed by a rapid build up of the slow phase and a normal duration.

In other subjects after a normal beginning there may be an early cessation of nystagmoid movement followed after some five or more seconds by a resumption of the normal rhythm (Fig. 15a). This inhibition may occur more than once in the course of an otherwise normal response (Fig. 15b), but the duration of nystagmus is not necessarily altered. Recognition of the possibility of this intermittent rhythm may prevent a period of inhibition being mistaken for cessation and an incorrect impression obtained.

Secondary Nystagmus. It has been shown that after rotatory stimulation the primary nystagmus is followed by a secondary nystagmus in the opposite direction. A similar reversed nystagmus is also seen after the primary one during optokinetic tests. The same

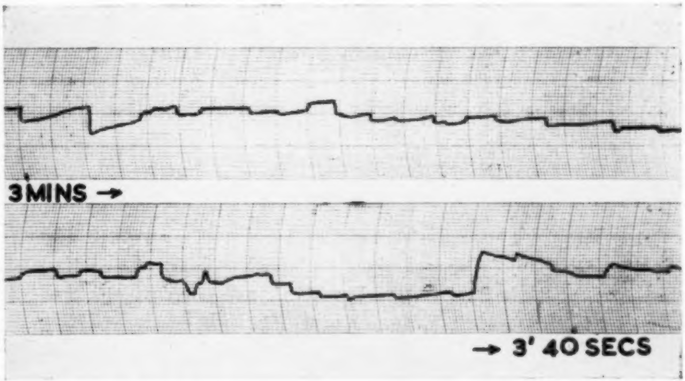

Fig. 16.—Onset of secondary nystagmus after caloric stimulation. Note the square waves during the transition period.

phenomenon can be observed in many subjects after caloric stimulation. Figure 16 shows the course of a nystagmus where the normal rhythm was replaced by a series of square waves for some forty seconds and then a secondary nystagmus in the opposite direction was ushered in by a brisk quick movement.

It is quite possible to mistake this secondary nystagmus for a persistence of the primary especially during visual observation of the eye movements, and to record a much longer response than is justified.

CONCLUSIONS

The existence of the anomalies in response in many apparently normal subjects may explain the occasional experience in testing patients that the results of caloric and other tests appear to be inconclusive or contradictory. The anomalies of inhibition and intermittence may suggest a very much shortened response and those of after nystagmus and secondary nystagmus may give an impression of protracted response. These anomalous phenomena have been noted, during visual observation of the eyes, in many patients during caloric testing, over many years. The apparent discrepancies were difficult to explain but the analysis, by electrical pickup and pen recorder, has clarified the various departures from normal. By electrical recording a very accurate estimation of the direction, speed, ampli-

tude and duration of all nystagmoid movements can be made and permanently recorded.

The square waves are an interesting variation and beside their own inherent interest are of value as indicating the end point of primary nystagmus and heralding the onset of secondary nystagmus.

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REFERENCE

1. Carmichael, E. A., Dix, M. R., and Hallpike, C. S.: Lesions of the Cerebral Hemispheres and Their Effects Upon Optokinetic and Caloric Nystagmus. Brain 77:345, 1954.

XXXVI

OBSERVATIONS ON PARALYSIS OF THE VOCAL CORDS IN SOME OF THE NEUROLOGICAL DISEASES

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Genoa, Italy

Observations on numerous neurological patients whom I examined in the course of over ten years of work as consulting physician in clinics for mental and nervous diseases, has permitted me to gather a considerable casuistics on laryngoplegia affections.

The problem of laryngoparalysis is probably one of the most studied and discussed in all otolaryngology, and the proof is given by the wide bibliography on the subject which cannot be summarized in a few lines, and which includes practically the most illustrious names who, since the dawn of the specialty to our days, have engaged themselves in clinical observations, experimental studies, histopathological investigations of the still open field of laryngoparalysis.

I shall recall, therefore, the by now classic works of: Auby, Baldenweck, Bellucci, Bilancioni, Mosella, Collet, Dejerine, Gavello, Lasagna, Masini, Massei, Mounier, Poli, Paget, Sterzi, Terracol, Waleminsky, Zigelman, up to the recent publications of Bondet, Brient, Caliceti, Carco, Collet, Fustenberg, Alajouanine, Lhermitte, Moore, Pallestrini, Pialoux, Pietrantoni, Piquet, Proctor, Sioberg, Stromwall, King, Cavanagh, Williams, Pirodda, Bellussi, Morrison, Van den Wildemberg, Woolman, De Vido, Maximov, Negus, Horbst, Clerf, Rustad, Del Bosque, Hofer.

In spite of the substantial contributions of research investigations, laryngoparalysis remains still an important subject for further studies.

This communication does not intend to discuss the various hypotheses, theories, or to test the results of the experimental, clinical, anatomical aspects of the subject under discussion. On the contrary,

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its main purpose is to report my personal observations as well as to draw the relative considerations.

Since 1946, I reported the following data regarding the examination of 76 neurological patients.

The analysis of data of the various neurological conditions summarized in Table I and the study of the type and frequency of the paralysis, sex and patients' age at the onset of the paralysis allow me

TABLE I

(Period 1942-1946)

NEUROLOGICAL DISEASE	NO. OF PATIENTS EXAMINED	NO. OF LARYNGOPARALYSIS	TYPE OF PARALYSIS	SEX	AGE
Pseudo bulbar paralysis	6	1	Bilateral of the abductors	M	58
Labioglossopharyngeal paralysis	5	2	a) Bilateral of the abductors and tensors	F	45
			b) Bilateral of the abductors	F	60
Multiple sclerosis	15	3	a) Right abductor	F	31
			b) Right abductor	M	39
			c) Left abductor	M	32
Syringomyelia	14	1	Left abductor	M	30
Tabes dorsalis	36	5	a) Bilateral of the abductors	M	43
			b) Left abductor and tensor	M	40
			c) Right abductor	M	62
			d) Bilateral of the abductors	M	41
			e) Right tensor	F	52

to confirm, in the majority of the morbid types, the results already reached by other authors.

Although the number of the studied cases does not allow me to draw definitive conclusion, my over-all observations are fundamentally in accordance with the majority of the investigators. For instance, as far as multiple sclerosis is concerned, I found motor laryngeal disorders in one-fifth of the patients who also presented the classical polymorphism of the disease process.

TABLE II

(Period 1947-1951)

NEUROLOGICAL DISEASE	NO. OF PATIENTS EXAMINED	NO. OF LARYN-GOPARALYSIS	TYPE OF PARALYSIS	SEX	AGE
Pseudo bulbar paralysis	4	—	—	—	—
Labioglossopharyngeal paralysis	4	1	Bilateral of the abductors	F	51
Multiple sclerosis	21	3	a) Bilateral of the abductors	M	42
			b) Left abductor	F	40
			c) Right tensor	F	30
Syringomyelia	19	3	a) Left abductor	M	35
			b) Right abductor and tensor	M	48
			c) Right abductor	F	41
Tabes dorsalis	48	4	a) Bilateral of the abductors	M	66
			b) Bilateral of the abductors	M	62
			c) Recurrent of the right	M	56
			d) Left abductor	F	60

Similarly, my observations on tabes dorsalis appear very close to those of Bilancioni, who found an incidence of about the 13 per cent of laryngoparalysis frequency in the tabetics, a figure very close to the 13.8 per cent which I have reported.

The majority of tabetic laryngoplegia incidence occurs between the age of 40 and 60 years and such period also in the majority of my cases (4 of 5) has shown to be the most affected by the same type of complication. As far as the localization of the paralysis is concerned, it is well confirmed, that in tabes dorsalis prevails by far the paralysis of the abductors and, with greater frequency, their bilateral paralysis.

With less frequency follows the recurrent complete paralysis, the paralysis of the constrictors, and the paralysis of other single muscles or muscular groups. Therefore, also under this point of view, my observations agreed with those of other authors.

The chapter of the labioglossopharyngeal paralysis alone has provided me with reports that, both from the point of view of the

TABLE III

(Period 1952-1956—First half year)

NEUROLOGICAL DISEASE	NO. OF PATIENTS EXAMINED	NO. OF LARYNGOPARALYSIS	TYPE OF PARALYSIS	SEX	AGE
Pseudo bulbar paralysis	3	--	----	----	----
Labioglossopharyngeal paralysis	4	--	----	----	—
Multiple sclerosis	18	2	a) Right tensor	F	42
			b) Bilateral of the abductors	M	32
Syringomyelia	19	3	a) Bilateral of the abductors	F	41
			b) Bilateral of the abductors	M	32
			c) Right abductor and tensor	F	38
Tabes dorsalis	32	1	Bilateral of the abductors	M	67

percentage (in labioglossopharyngeal paralysis the laryngoplegias are very rare) and from the point of view of the type of the paralysis (in the labioglossopharyngeal paralysis lesion of the tensor and of the abductors prevail) were contrasting with the existing knowledge in the literature.

Additonal observations on numerous neurological patients in subsequent years are briefly summarized in Tables II and III. In the former are included the cases observed from 1947 to 1951, and the latter corresponds to the 1952-1956 period (first half year).

The comparison of the findings of Tables I, II and III shows some evident percentage modification on the appearance of the laryngoplegia, which even with all due reservations suggested by the necessity of laregr statistical confirmation, seems to me worthy of some consideration.

The limited number of the studied cases do not permit me to draw any conclusion concerning pseudobulbar paralysis and the labioglossopharyngeal paralysis. Whereas in multiple sclerosis and in syringobulbia, it is noted a comparative constancy of reports which demonstrate a uniform percentage of laryngoplegias.

We would be inclined to conclude that most of the up to date therapeutic procedures used for the two above mentioned diseases were

of no help to modify the relation between the disease and its laryngeal affections. This may be better understood if we take into account etiological doubts and the consequent therapeutic uncertainties of these two neuropathological types.

On the contrary, various evaluations may be made on the subject of tabes dorsalis. The etiology of this condition is better known. Consequently there is a more widespread knowledge of the dangers linked with it, and of a more efficient social prevention.

In these latter years the appearance of antibiotics have further modified the situation in view of the fact that there has been a great diminution of the determining disease.

It is now acknowledged that the raditional clinical descriptions of many diseases have modified themselves due to the introduction of new types of therapy, with the consequent disappearance or modifications of old classical symptoms.

Confining myself to a few examples I would like to mention that tuberculosis of the larynx, one time so frequent, has presently almost completely disappeared. In the fields of lues there is no one who does not remember the frequency and the symptomatic effects of remarkable aneurism of the aorta which have become very rare nowadays.

In the light of such happenings it may not be surprising that even tabes dorsalis with laryngoplegia, which one time was a sign of a progressive trend of the morbid process, will become also, in consequence of the above mentioned new therapies, a more rare clinical entity.

These considerations make me believe that the progressive diminution of the percentage of laryngoplegias in tabes dorsalis, as demonstrated in my tables, may not be considered a casual fact but it represents the indication of a modification in the relation between the incidence of the occurrence of the disease and the concomitance of laryngeal symptoms, or it may be the expression of the decreased severity of the pathologic process.

SUMMARY AND CONCLUSIONS

In my report I attempted to present a brief review of the occurrence of laryngoplegias in pseudobulbar paralysis, labioglossopharyn-

geal paralysis, multiple sclerosis, syringomeylia and tabes dorsalis which I have observed during three periods of time between 1942 and 1956 (first half).

On the basis of my personal findings and those of the reviewed literature it seems to me that the frequency of the laryngoparalysis, in the above mentioned neurologic diseases, appears unvaried with the exception of tabes dorsalis which has shown a progressive reduction in incidence. This latter occurrence may be the result of more efficient social prophylaxis and particularly the introduction of the recently discovered therapies.

Piazza R. Rosetti, 28

XXXVII

CHRONIC OSTEITIS OF THE MAXILLA IN RELATION TO MAXILLARY SINUSITIS

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Estimations of the part which infection of the upper jaw (usually referred to as dental infection) plays in the causation of maxillary sinusitis have varied greatly with different writers and at different periods during the past half century.

Broadly speaking, the importance of this type of infection as a cause of sinusitis was grossly exaggerated fifty years ago, while now it is considerably underestimated. The writer considers that the term "dental" infection is a bad one, and believes that its use has been partly responsible for the present day lack of appreciation of the importance of this factor, for the following reasons: a) every untreated infection of a tooth or its supporting soft tissue ultimately leads to infection of the surrounding bone. From the point of view of the spread of sepsis, the infection of the bone is the important factor; b) not all infections of the jaws are due to infected teeth; c) residual chronic osteitis may remain in the jaw bone many years after the infected teeth which produced it have been removed. Many clinicians seeing an edentulous jaw regard it as being free of infection.

The writer has been deeply impressed by the frequency with which radiographs of apparently healthy edentulous jaws will reveal the presence of such conditions as retained tooth roots, chronic residual osteitis and infected cysts.

In order to investigate the effects which chronic infection in its various forms in the upper jaw adjacent to the antrum might have on that cavity, 52 cases have been studied as follows:

In 4 cases specimens were obtained from a public hospital where, during the extraction of a tooth with peri-apical infection, part of

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the antral wall with its mucosa attached had come away with the tooth.

In the remaining 48 cases, the following procedure was carried out: a mucoperiosteal flap was retracted to expose the septic area in the jaw; any infected tissue present was removed. The antrum cavity was then entered and a specimen of its mucosa near the infected bone was removed for biopsy.

In 26 cases an opening through the bony wall of the antrum was found. In some it was quite large, in the majority it was small and had to be enlarged in order to gain access to the antrum cavity. In 22 cases there was no opening and a small window was cut through the bone.

Clinically, some of the patients presented signs and symptoms of chronic sinusitis, others did not. Most of the patients in the latter class had been first seen by a dentist who had discovered the infection because of the patient's complaint of tenderness of the jaw, or on routine radiographic survey of the jaws.

A striking and unexpected result of this investigation has been that in every case pathological changes have been found in the antral mucosa, and that a considerable majority of them have been of a gross nature. In all cases the mucosa was thickened and in some of them pus was present. In a large proportion, the pathological changes were more pronounced in, or were confined to, the base of the sinus.

Histopathologically, a sclerosing fibrotic type of change predominated in 36 cases; in 11 a hypertropic type of sinusitis was found, in 2 cases sclerosing and hypertrophic changes were about equal. Two cases showed a partly sclerosing and partly polypoidal sinusitis, and in one case large areas of gland tissue were found. Cell infiltration was chiefly with small round cells and plasma cells, but some showed large numbers of neutrophile polymorphonuclear leucocytes. In two cases there were numerous eosinophiles. In a few cases the epithelium was intact; in most the cilia had been destroyed; in some only patches of basal epithelial cells remained, and in others there was not trace of any epithelium.

This description of the research carried out is a brief summary of the investigation, which was presented in 1954 as a thesis for the diploma of laryngology and otology. In this work, which contains

full pathological details of the cases studied, the unsatisfactory nature of classifications of the types of sinusitis is pointed out. Probably the widest used is that of Eggston and Woolf.[1] Their classification is as follows:

1. Hypertrophic, or polypoidal sinusitis
2. Sclerosing, fibrotic or arteriofibrotic sinusitis (atrophic)
3. Papillary sinusitis
4. Follicular sinusitis
5. Glandular or adenomatous sinusitis

Sclerosing sinusitis is described as "dry." In the writer's experience, many grossly hypertrophic antrum linings are not polypoidal, but have a high content of granulation tissue, and many sclerosing or fibrotic cases are neither atrophic nor dry. Other classifications are even less satisfactory than that of Eggston and Woolf.

Most cases were characterized by a varied histological picture when sections were taken from different areas, and the most that could be said was that one or other type of pathological change predominated.

Management of these cases will be discussed under the following headings:

1. Radiographic technique
2. Co-operation with the dental profession
3. Surgical treatment.

Many of these cases are not diagnosed because of unsatisfactory radiography, or lack of co-operation between the rhinologist and the dentist.

RADIOGRAPHY

Anteroposterior radiographs of the sinuses, while indispensable for giving a view of the antrum as a whole, and a comparison of the two sides, dot not show in clear detail the floor of the cavity which is also the alveolar process of the maxilla. Lateral head radiographs are sometimes useful, but the best pictures of this area are obtained by the small 4 x 3 cm intra-oral films, usually known as "dental" films. No other radiographic technique is as reliable for revealing the various

types of infection which are found in the jaws. The commonest of these are: teeth with periapical and periodontal infection, retained tooth roots, chronic residual osteitis and infected cysts.

At the same time, these small intraoral films give valuable information of changes in the antrum floor which are often completely missed by anteroposterior radiography. For example, breaks in the bony floor, tooth roots within the antrum cavity, localized areas of swollen mucosa confined to the floor.

CO-OPERATION WITH THE DENTIST

In the diagnosis of sinusitis associated with infection of the maxilla, careful clinical examination of the teeth and jaw bone should be carried out in addition to adequate radiography. For the rhinologist who has not had any training in dentistry, this necessitates close co-operation with the patient's dentist (often advocated but not so often carried out). Members of the dental profession should be made aware of the seriousness of chronic sinusitis and of the direct bearing which septic conditions in the maxilla have on its production.

For many years the writer has been opposed to the retention of pulpless teeth in close relationship to the antrum. This opinion was criticized by the editor of the "Year Book of Dentistry" of 1953[2] but further recent discussions with various Australian, American and English authorities on the reliability of modern endodontic treatment in eliminating sepsis in every case have only confirmed this opinion. Even in the most skilled hands this treatment is not 100 per cent efficient, and it is too expensive to be universally used. Partly because of this and partly from the lack of the necessary skill, the endodontic work of many dentists throughout the world is not in accordance with the best modern principles and therefore is not satisfactory or safe as far as the antrum is concerned.

SURGICAL TREATMENT

In every case of maxillary sinusitis caused by, or complicated by, infection of the alveolar bone, treatment should be complete eradication of the jaw infection, combined with a) antrum wash-outs until a clear return has been obtained, or b) antrostomy with removal of the infected antral mucosa. Antrum wash-outs following jaw surgery have a reasonable chance of success in those cases in which there are only slight mucosal changes in the antrum. Cases with moderate or

extensive mucosal swelling should be treated by antrostomy. Sometimes, if the infection has been of long standing, or coupled with nasal infection, a radical antrum may be necessary. However, it has been the experience of the writer that, in the vast majority of cases of sinusitis associated with jaw infection, only the mucosa of the floor and possibly the lower parts of the other walls will be infected. In such cases the upper part of the antrum should not be disturbed.

Sometimes the jaw infection may be dealt with by the dentist prior to any antrum surgery, especially in cases of periapical or periodontal infection requiring only simple teeth extraction. However, the writer considers that, if the jaw infection is such that it requires the turning back of a mucoperiosteal flap, this should be done at the same time as the antrostomy, and is quite within the province of the ear, nose, and throat specialist.

This procedure almost invariably requires a lower incision than the usual Caldwell-Luc incision; the flap is longer and more awkard to retract, but the advtanges are that the incision is over solid bone, repair is rapid, and the risk of a postoperative oro-antal fistula almost nonexistent. Location of tooth roots is sometimes difficult and may require special radiographic technique. The bone surrounding any buried tooth root should be considered as being infected whatever the radiographic appearance, and should be thoroughly curetted and the rough edges trimmed smooth.

Chronic osteitis of the jaws is chiefly a rarefying process. Sometimes a definite cavity, almost always larger than is indicated by the radiograph, is found, containing some or all of the following: pus, granulation tissue, tooth fragments, small sequestra. Sometimes the trabeculae have not completely broken down and the marrow spaces are occupied by chronic inflammatory tissue.

These areas should be curetted until sound bone is reached. Mutilating bone destruction is not necessary. In an article published in 1952[3] Bedford-Russell and Wright gave a list of the aims of the operation in these cases. Two of their aims were: the complete eradication of all alveolar cancellous bone, and the removal of diseased bone of the antral wall in part, or wholly. Such drastic treatment is not only unnecessary, but is harmful to the patient.

In the case of infected cysts of the jaw, small ones which do not encroach much on the antrum cavity may be shelled out before

the antrum is opened, but in the case of large cysts, it is usually necessary to break down the intervening wall and make one cavity of cyst plus antrum.

In very septic cases, an antronasal window is made, but if no pus is encountered and a good natural ostium exists, this is often unnecessary.

The writer has performed several hundred of these operations in addition to the 52 cases which were studied from the angle of their pathology. In almost all of these the results have been most satisfactory.

61 Collins St.

The writer wishes to express his appreciation of the co-operation of Dr. T. F. Coffier, clinical photographer, Victorian Eye and Ear Hospital, Melbourne, who was responsible for most of the illustrations.

REFERENCES

1. Eggston, A. A., and Woolf, D.: Histo-Pathology of the Ear, Nose and Throat. Williams and Wilkins Company, Baltimore, p. 629, 1947.

2. Editor's Note: Year Book of Dentistry, p. 311, 1953-1954 Series.

3. Bedford Russell H. G., and Wright, Leslie: The Significance of Alveolar Osteitis in Rhinology. Proc. Royal Soc. of Med. 45:12 (Dec.) 1952.

XXXVIII

REPORT OF A DEATH FOLLOWING ACUTE OTITIS MEDIA

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There can be little doubt that the use of antibiotics and chemotherapeutic agents has been a major factor in the decrease in the morbidity and occasional mortality resulting from otitis media and its intracranial complications during the past 20 years since 1937 when the sulfonamides were first used and since the early 1940's with its antibiotic medication.

In this era of spectacular results from antibiotics and chemotherapy in acute otitis media, one seldom thinks of the possibility of sudden death as the result of intracranial complications. The case to be reported demonstrates that this is still possible. The literature of presulfonamide and antibiotic time is replete with reports illustrating cases of spread of infection from otitis media by extension through vascular channels. Eagleton's index,[1] published in 1941, a monumental compilation of abstracts of the progress of disease in intracranial lesions related to aural conditions, is filled with such cases. The case to be reported involved desperate measures for an extreme situation. For an otologist practicing in a small community to lose a patient with a relatively simple ear infection is a severe shock and so infrequent these days that the writer is prompted to report it in full.

REPORT OF A CASE

Mr. R. P., a 39 year old white male, was first seen on December 26, 1955, with a complaint of a stuffy feeling in his left ear for a few days. He was found to have a secretory otitis media. Paracentesis produced thick syrupy fluid removed by inflation. His hearing was normal a few days later and the drum was healed. No antibiotics were given. The patient was not seen until the morning of February 11, six weeks later, when he stated he had developed a very severe earache the night before. The left tympanic membrane was bulging

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and was incised releasing considerable pus. Temperature was 99.2. He was given erythromycin 250 mg every six hours. He had more pain in the evening. About 4 A. M. on February 12 his wife called and stated that he had a high fever, was delirious and unable to speak clearly. Examination at the home showed this to be true and in addition he had neck rigidity, rotary movements of the eyes, bilateral proptosis and a positive Kernig's sign. He was taken at once to the hospital.

Hospital Admission Note. A preliminary diagnosis of meningitis was made. Temperature was 103.6, pulse 110, respiration 48. Patient was a very obese individual, height 73 inches, weighing 255 pounds. He was in a very violent convulsive state and completely irrational. He required the combined efforts of six people to restrain him. One million units of penicillin and 0.5 grams of streptomycin were given. About two and one-half hours later spinal puncture produced cloudy fluid under considerable pressure which showed a large number of neutrophiles and groups of gram positive encapsulated diplococci. Cultures from blood and spinal fluid showed no growth after 48 hours probably as a result of antibiotic therapy. He was given another million units of penicillin intraspinally and then an intravenous injection of 5 grams of sulfadiazine in 1,000 cc of saline. A little while later one million units of penicillin were given intramuscularly. His downhill course was very rapid. He became more violent, convulsive and cyanotic. Respirations were very rough and irregular. At 11:30 A.M. he died.

GROSS AUTOPSY REPORT

Upon examination of the head, the scalp and calvarium are normal. There is approximately 100 cc of liquid and clotted blood present in the subdural space surrounding almost the entire brain. The external surface of the brain shows a purulent meningitis with purulent exudate present. Cut sections of the brain show nothing of note aside from the purulent meningitis. Examination of the base of the skull reveals complete thrombosis of the left lateral sinus as well as the inferior and superior petrosal sinuses. There is also thrombosis of the right lateral sinus and a portion of the superior longitudinal sinus. The tympanic portion of the left temporal bone shows no gross changes.

MICROSCOPIC AUTOPSY REPORT[2]

In the mastoid portion there is a chronic osteomyelitis, the spaces being occupied by rather old granulation tissue. There is an intense

chronic suppurative inflammation in the attic. The coils of the cochlea included in the sections show intense chronic inflammation.

This is evidently a long standing suppurative otitis media that has pursued a not uncommon course in untreated cases with involvement of the attic and the cancellate and pneumatized portions of the mastoid and the petrous.

In the relatively short course of the final fatal termination of this case, which seemed to be 24 to 36 hours, it is very difficult to evaluate the onset of the sinus thrombosis on the one hand and the meningitis on the other. It takes some time to develop an exudate in meningitis so one might suspect that both of these conditions must have been present for a few days at least prior to the fatal fulminating convulsive and comatose stage which terminated in death. Most of the otologists to whom the writer submitted the history and findings felt that there was no connection between the simple secretory ear which occurred six weeks previously and the sudden development of the new complicated picture. Hoople,[3] however, stated that in the preantibiotic era he has seen two or three similar cases when death occurred in 24 hours. House[4] felt that the thrombosis developed rather slowly by retrograde parathrombophlebitis and when it broke through into the meninges symptoms developed very rapidly. Then add the effect of the extreme bilateral thrombosis which would be worse than a bilateral jugular ligation plus the violent convulsive struggles and the impossibility of survival becomes only too apparent. No vascular bed can stand such an attack for long. Rupture of the lateral and petrosal sinuses was inevitable since the blood had no other place to go. The writer leans toward the point of view that the thrombosis developed before the meningitis. Eagleton[1] in his abstract of intracranial lesions related to aural conditions made a great contribution to otology. In 1926 he stated that the condition still continued to be associated with a mortality of nearly 100 per cent. He also stated that the earlier the beginning of meningitis is recognized and the earlier the inflammatory process is removed the better are the prospects of a cure. Eagleton mentioned the deceptive character of otitis media following infections by streptococcus mucosus. He cited Hlavacek of Hungary who reported a case of sinus thrombosis and meningitis with only slight changes in the tympanic membrane. Eagleton also felt that the complications were more often a matter of virulent activity of the organism than the kind of organism. Courville[5] surveyed autopsy findings in the Los Angeles area for otitic complications comparing the years 1928 to 1933 with 1949 to 1954. He found such complications had been reduced by 90 per cent but

still occurred in variable forms at a rate of approximately 25 in every 10,000 deaths. All were meningitis cases while dural sinus thrombosis was completely eliminated. Nielson[5] in the same report noted that antibiotics have reduced the number of intracranial complications but have made the diagnosis more difficult. Careful observation and constant vigil are still of extreme importance lest the physician be deluded by the apparent innocuous nature of the course of events.

A very recent report of Rosenwasser and Adelman[6] from the otolaryngological service of the Mount Sinai Hospital stresses the increasing frequency of otitic complications. Inadequate dosage and improper choice of antibiotics plus discontinuing the therapy too soon are mentioned as causes of these conditions following middle ear disease.

SUMMARY

A case of acute otitis media has been presented, terminating in a sudden fatal onset of violent convulsive meningitis and dural sinus thrombosis. A partial review of the literature reveals that in spite of watchful care and antibiotic therapy these dreaded sequelae can still occur. As otologists we must occasionally expect to see these infrequent cases and treat them with the best means at our command.

62 East 4th St.

REFERENCES

1. Eagleton, W. P.: Eagleton's Index. Abstracts of Progress in Intracranial Lesions Related to Aural and Nasal Conditions, 1941.

2. Ash, J. E.: Personal Communication to the Author.

3. Hoople, Gordon D.: Personal Communication to the Author.

4. House, Howard P.: Personal Communication to the Author.

5. Courville, Cyril B.: Intracranial Complications of Otitis Media and Mastoiditis in Antibiotic Era. Laryngoscope 65:31-46, 1955.

6. Rosenwasser, H., and Adelman, N.: Otitic Complications. A.M.A. Arch. Otolaryng. 65:225-234, 1957.

The Scientific Papers of the American Otological Society

XXXIX

MICROELECTRODE STUDIES ON THE AUDITORY NERVOUS SYSTEM

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Since experiments using microelectrodes for examining responses in the auditory nervous system have been under way for about 15 years, a survey of the results thus far achieved may be in order. The auditory structures that have been examined include the auditory nerve, cochlear nuclei, superior olive, inferior colliculus, medial geniculate and auditory cortex. At least some effort has therefore been devoted to each of the main stations of the classical afferent pathway. The major preoccupation of the experimenters has been with the problem of how frequency of tonal stimulation is mediated in the nervous system, but loudness and the consequences of stimulating both ears together have received attention also.

The goal of microelectrode experiments is to isolate a single brain cell for study. The way this cell reacts to different sounds is established during life and its position in the brain is approximated post mortem. By the judicious integration of the physiological and anatomical data so obtained a concept of how the brain reacts to sound stimuli is synthesized.

Auditory units ordinarily show reasonably specific responses to sounds. Thus tones may set units into activity (excitation), or stop the activity then in progress (inhibition). If excited by tones, a unit will be aroused at its threshold by a limited band of frequencies, and this band is called its characteristic frequency. In a similar manner

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only certain tones will inhibit a unit if this happens at all. It turns out that throughout the brain most units are indifferent to some tones, but that all heard tones will activate some units specifically.

In the dorsal cochlear nucleus, for example, characteristic frequencies vary systematically from high at the medial (dorsal) edge to low at the lateral (ventral) margin. Limited data concerning both the posteroventral and anteroventral cochlear nuclei suggest that all frequencies are represented in each, with highs located dorsomedial and low frequencies probably ventrolateral. When an electrode passes successively through all three nuclei, there first occurs an orderly progressive shift of characteristic frequencies corresponding to successive advance through the dorsal nucleus. A sudden, large change in characteristic frequency takes place upon penetration of the posteroventral nucleus and a new orderly sequence starts. A third large change occurs upon entry into the anteroventral nucleus followed by a final sequence of shifts in characteristic frequencies as this nucleus is traversed. Since a physiological counterpart for the known anatomical "unrolling" of the cochlea exists at this level within the central nervous system, it may be concluded that one of the correlates for pitch is the place in the brain where the tone produces its effects.

In the superior olive, single units have been isolated in all of its five major subdivisions, and these may be driven by either ear or by both. With respect to tones, the units found here behave in a manner suggesting that they mainly transmit the analysis performed at the cochlear nucleus. The accessory segment is of particular interest in another connection for units here behave significantly differently when diotic time differences of the order of 0.1 msec are introduced. Since in man binaural localization of sounds occurs when time differences of just this magnitude are involved, the unit studies here appear clearly related to this important auditory psychological function.

In the auditory nerve, inferior colliculus, medial geniculate body and cerebral cortex the unit responses, so far as they have been studied, support the following statements which are offered here tentatively as some of the principles by which the nervous system mediates auditory information.

1. The basilar membrane of the cochlea is "unrolled," both anatomically and physiologically, at the various levels of the classical

afferent pathway. A particular tone, therefore, produces its effects at particular places in the brain. Another tone will do the same, but at different places.

2. The neural effects of each sound will include both excitation and inhibition, and every sound, whether it be tone, click, or noise, probably produces a unique pattern of excitation and inhibition in the cells of the brain.

3. When both ears are stimulated some units are exquisitely sensitive to whether or not the sounds have been presented simultaneously. Time differences of one hundred microseconds between stimuli can clearly be preserved in terms of unit responses at some brain locations.

It will be freely confessed by those who work with microelectrodes on the auditory system that the technique has certain limitations. These limitations stand out especially when one considers the so-called higher auditory functions like learning to respond to sounds and paying attention to some sounds and not to others. The possibility that the microelectrode technique may prove of value in investigating such problems improves as the art progresses, but regardless of this consideration a continued useful future for microelectrode studies on the auditory system can be confidently predicted.

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XL

BEHAVIORAL STUDIES OF AUDITORY DISCRIMINATION

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Chicago, Ill.

There has been a shift in emphasis of research on hearing. The mechanism of cochlear analysis of sound has ceased to be the primary concern of investigators; they have turned their attention more and more to the problem of how the patterns of activity aroused in the cochlea by peripheral stimulation are transformed into nerve impulse messages and to the role of the central nervous system in transmitting and decoding these messages.

Dr. Rosenblith and Dr. Galambos have shown how methods of the biophysicist and electrophysiologist may be utilized to further our understanding of the neurophysiology of hearing. I shall try to show the kind of contribution that can be made by the psychophysiologist.

The aim of the psychophysiologist is to manipulate physiological conditions and to measure changes in behavior of an experimental animal. To accomplish this aim, he borrows methods of training and testing animals from the comparative psychologist; techniques of experimental surgery from the physiologist; and, in order to maximize the information he is to get from his experiment, some of the techniques of the neuroanatomist and electrophysiologist. The methods used and the results obtained in a number of psychophysiological studies will be described briefly. The purpose of these studies was to discover the role played by the auditory cortex in auditory discrimination.

The cortical projection areas of the auditory nervous system have been defined in a number of ways: by cytoarchitectonics, by

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tracing degenerating pathways to or from thalamic nuclei, and by recording of evoked potentials when the ear is stimulated by sound or the peripheral nerve is excited by electric shock. The maps of auditory cortex obtained by these different procedures are not exactly alike although there is reasonable agreement. At the time we began the experiments which I shall report, we chose the evoked potential maps as most nearly representing the total region of cortex receiving the input of the main auditory afferent system. The evoked potential map for the cat, the animal used in our experiments, is shown in Figure 1. This map is based upon the results of a number of studies, particularly those of Woolsey and Walzl,[8] of Rose and Woolsey,[7] and of Hind.[4]

Two procedures were employed in the training and testing of animals. To measure ability to localize sound in space, the apparatus pictured in Figure 2 was used. The cat was placed in a starting cage; a buzzer was sounded behind one of the food boxes located along the perimeter of the semicircular enclosure; the cat was released and allowed to approach, open the door, and obtain food from the box behind which the buzzer was sounded. If it went to the other box, it was not rewarded and was returned to the starting cage for another trial. The angle between the two boxes and, thus, between positions in which the buzzer was sounded could be varied from 0° to 180°.

To measure frequency discrimination and pattern discrimination in the cat, a box consisting of two compartments was used. The floor and walls of each compartment were made of one-quarter inch brass rods spaced about three-quarters of an inch apart. Electric current could be passed through these grills. The experimental animal was placed in one of the compartments of the double-grill box; a pulsing tone was sounded (Fig. 3); at a change in the frequency of the pulsing tone or in the pattern of a sequence of tones, shock was given until the animal crossed from one compartment to the other of the box; shock was avoided by the animal when it learned to cross at the time of the shift from negative to positive stimulus.

After training in one or more of these auditory discriminations, all or parts of the auditory cortex of experimental animals were ablated. Ablations were done under aseptic conditions; subpial suction was used to remove cortical tissue. After a recovery period of

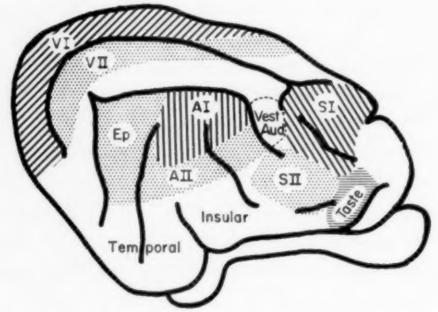

Fig. 1.—Diagram showing sensory projection areas of the cerebral cortex of the cat.

approximately two weeks, animals were returned to the discrimination situation for tests of retention of the learned habit and for retraining if necessary.

At the end of postoperative training, the cortex surrounding the areas ablated was explored by electrophysiological methods in some animals. In this way, small remnants of active auditory cortex, i.e., cortex from which evoked potentials can be recorded, may often be detected. The brains of all animals were sectioned and stained. From the serial sections, cortical lesions were reconstructed and thalamic degeneration was mapped.

Results of several studies which have been completed and reported elsewhere may be summarized briefly:

1. After large bilateral lesions of auditory cortex, cats show a deficit in ability to localize sound in space. Whereas normal animals can make correct responses 75 per cent of the time at angles as small as 5°, the operated animals make similar scores only at 40°. Furthermore, the animals with auditory cortex damage suffer some decrement in performance, even at angles as great as 180°.[5,6]

2. Animals with complete bilateral ablation of auditory areas I, II, and Ep show little or no loss in capacity to discriminate small

changes in frequency of pure tones. The learned habit is lost following ablation but is relearned at about the same rate as original learning. The temporary loss is specific to the auditory habit; behavior indicating general adaptation to the test situation is unchanged.[1]

3. Animals with complete bilateral ablation of auditory areas I, II, and Ep show loss of capacity to discriminate changes in the temporal patterning of tones. If a small remnant of these areas is left intact, relearning of the discrimination occurs; with complete ablation, relearning is not possible.[2]

During the progress of these experiments on localization of sound in space, frequency discrimination, and pattern discrimination, it became increasingly apparent that the information we had from anatomical studies of thalamocortical relationships of the auditory system was incomplete. In our experiments, we found that ablation of AI led to severe degeneration of the anterior half of the principal part of the medical geniculate body. When AI, AII, and Ep were ablated, approximately two-thirds of the principal part of the medial geniculate degenerated. However, no degeneration of the geniculate was observed after lesions confined to AII or Ep.[1,2,6] These results confirm those reported earlier by Rose and Woolsey.[7] In all cases of lesions which did not extend far beyond the boundaries of AI, AII, and Ep, the posterior tip of the medial geniculate remained intact.

In some cases, particularly in the experiment on pattern discrimination, the cortical ablation was intentionally made larger so that it included cortex ventral to AII and Ep. In these cases, it was observed that retrograde degeneration in the thalamus extended further into the posterior region of the principal division of the medial geniculate and invaded the magnocellular division.

In an anatomical study now in progress, Dr. K. L. Chow, Dr. I. T. Diamond, and I have made lesions of the temporal and insular cortex ventral to AII and Ep. After these lesions, retrograde degeneration can be observed in the posterior part of the medial geniculate. The degeneration is less severe than that seen in the anterior medial geniculate after AI lesions but, in our opinion, it indicates that there is projection from the posterior tip of the medial geniculate to the cortex ventral to AII and Ep.

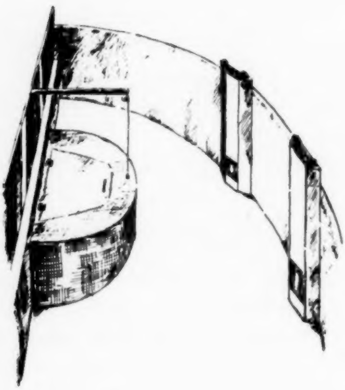

Fig. 2.—Sketch of apparatus used in testing localization of sound in space. Only part of the semicircular enclosure is shown. The starting box is on the left; food boxes are shown on the right. The experimental animal is trained to go to the food box behind which a buzzer has been sounded.

These anatomical findings have led us to new behavioral studies. If we define the auditory projection region of the cortex as that region, the ablation of which leads to complete degeneration of the medial geniculate body, then our maps of auditory cortex must be altered to include part or all of the area lying between the ventral boundary of AII-Ep and the rhinal fissure. We are now doing experiments to discover the effects of ablating this more extensive region. The results of these studies are not ready to report as yet, but I do want to mention a rather striking behavioral loss we have observed after lesions confined to this "ventral auditory area."

In a study being done with Mr. J. M. Goldberg and Dr. Diamond,[3] we have found that bilateral ablation of the ventral area alone produces profound loss of ability to discriminate changes in tonal patterns. The learned discriminatory habit is not reacquired after prolonged retraining. A frequency discrimination can be relearned. When the cortex of these animals was explored at the time they were sacrificed, good evoked potentials were obtained from AI, AII, and Ep. To the best of my knowledge, this is the first instance in which

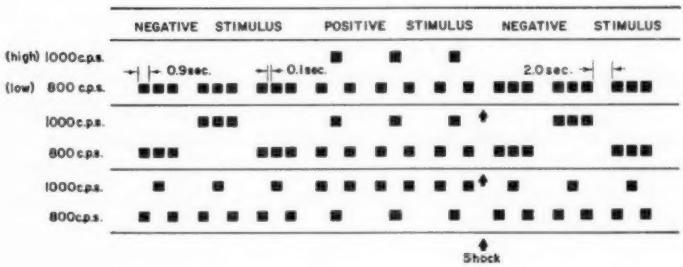

Fig. 3.—Diagram to show sequences of tones used in frequency and pattern discrimination. The sequence illustrated by the top line requires only discrimination of frequency. The negative stimulus is a pulsing 800 c.p.s. tone; the experimental animal is trained to respond when a new frequency of 1000 c.p.s. is added. In the sequences illustrated by the middle and bottom lines, the discrimination has to be made on the basis of a change in temporal patterning.

a severe and lasting deficit in auditory discrimination capacity has been produced in an animal by a cortical lesion which did not include all or nearly all of the primary projection areas. That this ventral area is not simply additional auditory projection cortex seems clear. In the total complex sensory-motor circuit involved in auditory discrimination, the ventral area may lie in series with other parts of the auditory cortex so that with its destruction the integration of activity taking place in the other sectors is blocked or distorted in its transmission. Another hypothesis is that the ventral area lies in parallel with other areas of the auditory cortex and that its ablation in some manner alters the normal course of events in the remaining intact part of the geniculocortical circuit.

Our speculations about the function of the ventral area and about the neural mechanisms of auditory discrimination must remain rather vague (and probably wrong) until we have considerably more evidence of several varieties. We need more exact information about both afferent and efferent, cortical and subcortical connections of the different auditory areas. From electrophysiological studies, we need data about the interaction of the main auditory system with other systems which we know are important in control of cortical function,

e.g., the ascending reticular and the limbic systems. Finally, we need to increase the subtlety of our methods of testing auditory discrimination and the exactness of our ablation techniques.

I have not attempted, here, to relate the results of our experimental studies to those of clinical studies of neural disorders of hearing. In our experiments, we are seeking to understand the neurophysiological mechanisms of auditory discrimination. Through the use of anatomical and electrophysiological methods, we try to define neural units, e.g., the geniculocortical part of the auditory system. We then change or destroy this unit and measure the effects upon discriminatory behavior. In order that we may find a pronounced deficit, it is usually necessary that we completely destroy the given unit bilaterally if it is a part of a symmetrically bilateral system. Having found changes which occur after ablation of large units, we can go on to smaller units. I have given an example of our attempt to do this.

In the clinic, in contrast to the experimental laboratory, the problem is usually not one of discovering neural mechanisms but one of diagnosis, of measuring an auditory deficiency, and attempting to specify the locus of a lesion or diseased condition. The lesion will almost never be confined to or include all of an anatomically defined unit; it will usually be unilateral.

At the present stage of research on neurophysiology of hearing, the findings of the investigator in the experimental laboratory may be suggestive to the clinician; we can hardly hope for more. As scientists, I think we can all agree, however, that in the long run the best diagnosis and treatment of disorders of hearing will be attained when we have gained an adequate understanding of the basic physiological mechanisms involved.

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REFERENCES

1. Butler, R. A., Diamond, I. T., and Neff, W. D.: Role of Auditory Cortex in Discrimination of Changes in Frequency. Jour. Neurophysiol. 20:108, 1957.

2. Diamond, I. T., and Neff, W. D.: Ablation of Temporal Cortex and Discrimination of Auditory Patterns. Jour. Neurophysiol. 20:300, 1957.

3. Goldberg, J. M., Diamond, I. T., and Neff, W. D.: Auditory Discrimination after Ablation of Temporal and Insular Cortex in Cat. Fed. Proc. Am. Soc. Exp. Biol. 47:204 (abstr.) 1957.

4. Hind, J. E.: An Electrophysiological Determination of Tonotopic Organization in Auditory Cortex of Cat. Jour. Neurophysiol. 16:475, 1953.

5. Neff, W. D., Arnott, G. P., and Fisher, J. F.: Function of the Auditory Cortex: Localization of Sound in Space. Am. Jour. Physiol. 163:739 (abstr.) 1950.

6. Neff, W. D., Fisher, J. F., Diamond, I. T., and Yela, M.: Role of Auditory Cortex in Discrimination Requiring Localization of Sound in Space. Jour. Neurophysiol. 19:500, 1956.

7. Rose, J. E., and Woolsey, C. N.: The Relations of Thalamic Connections, Cellular Structure and Evocable Electrical Activity in the Auditory Region of the Cat. Jour. Comp. Neurol. 91:441, 1949.

8. Woolsey, C. N., and Walzl, E. M.: Topical Projection of Nerve Fibers from Local Regions of the Cochlea to the Cerebral Cortex of the Cat. Johns Hopkins Hosp. Bull. 71:315, 1942.

XLI

ADVENTURES WITH THE STAPES

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LONDON, ENGLAND

Once again the stapes has become the center of attraction to all who are interested in deafness and I would like to tell you of my experiences with the fascinating, though fragile, little bone.

I hope that you will forgive me if I use the personal pronoun from time to time, as this is a personal account of adventures with the stapes; though before I have finished the thought may occur to some of you that a more appropriate title for this talk would be "Misadventures with the Stapes."

My interest in the stapes and in otosclerosis was first aroused when I was House Surgeon to Cheatle and Jenkins in 1926. Cheatle taught me, among many other things, how to tell to which side a stapes belonged and by this time Jenkins had abandoned the idea of surgery for otosclerosis owing to repeated disappointments, and he told us very little about the opening of the lateral semicircular canal which he had described first in 1913.

In 1935 I listened to Sourdille describe his tympanopexy and in 1938 I paid a visit to Gunnar Holmgren in Stockholm. After reading all that I could about the new fenestration operation described by Julius Lempert I tried to do it; but without success. After the war I started again but still did not make much headway. This was largely due to imperfections in technique, but also to some extent to the advanced deafness of those patients willing at that time to submit to surgery at my hands.

Having read of the work of Kessel and later of Jack on stapes removal I decided to give this procedure a trial. Using the conventional fenestration approach as described by Lempert I set out to remove the stapes. At once I discovered that in otosclerosis this bone

was very fragile and in consequence far from easy to remove; though I often found that it was not so firmly fixed as I had been led to believe. Between the autumn of 1945 and the early summer of 1946 I removed, I am afraid to say piecemeal, the stapes from 14 patients, all with otosclerosis. After removal of the stapes I placed a small piece of amniotic membrane over the oval window and then pressed the tympanomeatal flap into the oval window holding it in position with a plug of bone wax. In two patients only was there any improvement in hearing; and one of these has maintained his improvement to this day. In fact this patient who had his stapes removed in 1946 had a successful fenestration on the other ear in 1949. I must add however that the hearing improvement is better on the fenestrated side.

I am thankful to be able to record that in none of the others was the hearing made worse; though I must admit that in some there was little room for deterioration. I was always interested to find how deeply set the footplate was in the oval window, and how the anterior crus of the stapes seemed to be gripped by the overgrown margins of the oval window in front. In 1938, I had already had the opportunity of seeing the stapes in cases of Ménière's disease under the Leitz binocular dissecting microscope giving ten diameters of magnification. In these cases it was interesting to find that the stapes moved freely, and in particular that the anterior crus was quite free; and the rounded anterior edge of the stapes footplate could be seen as a distinct ridge through the front end of the annular ligament when light pressure in a backward direction was applied to the neck of the stapes. Later when I started operating upon patients with otosclerosis I found that the stapes particularly at its anterior end looked quite different. The anterior crus was not free and the movement of the anterior end of the stapes footplate could not be seen. That is not to say that the stapes was immobile. There was usually some movement there though it was obviously restricted. I soon found out how easy it was with a stiffened stapes for the crura to break. In fact in these cases removal of the stapes could only be carried out by first of all pushing the footplate in. It nearly always broke across and the fragments had to be removed from inside the vestibule by using two fine dental reamers as Chinese chopsticks to pick them out.

Now when I was invited by the Triological Society to their meeting in Chicago in 1946 I naturally took the opportunity of

seeing Julius Lempert at work. I must confess that I arrived in New York in a somewhat biased frame of mind. I had read Lempert's description of his operation carefully, and I thought that I understood it. I had tried it out but had not been able to achieve any success. In my innocence, or perhaps I should say conceit, I felt that possibly the method was at fault. However, another Julius, I refer to Julius Caesar, hit the nail on the head when Shakespeare made him say, "The fault, dear Brutus, lies not in our stars but in ourselves." Continuing the analogy, and it is not inappropriate as this year is the 2,000th anniversary of the Ides of March, I would like to paraphrase another famous saying of Julius Caesar to suit the situation, "I came, I saw, I was conquered." For I soon realized that I had a lot to learn about fenestration and Julius Lempert did his best to improve my otological education, and for this I shall always be grateful to him.

On my return to England I reread his writings and with what I had learned from watching him I was at last able to profit by what he had written, and found that I was able to get good results with the fenestration operation.

About 1950 I started another series of stapes removal, still using the Lempert approach but removing the whole of the malleus so that the tympanic membrane could more easily be displaced inwards so as to cover over the oval window. There was some improvement in the hearing of five out of eight patients operated upon in this way. However, the gain in hearing was not sufficient to justify continuing this procedure, and so I soon reverted to the fenestration operation which gave consistently better results.

About this time I was operating on some very advanced cases of otosclerosis and in some I did a fenestration of the lateral canal as well as removal of the stapes. I was, however, sharply reminded of the folly of this procedure by one patient who developed a Tullio phenomenon, that is to say, he became very giddy each time a loud sound reached his ear. This was so distressing to him that eventually I had to destroy his labyrinth. I have seen three other patients with disabling vertigo after having been submitted to a fenestration operation in the presence of a mobile stapes.

The reason for this is quite simple when we study the diagrams introduced by Wever and Lawrence.

As a result of these experiences I came to the conclusion that the Tullio phenomenon depends upon the existence of two mobile windows on the vestibular side of the labyrinth. Consequently I feel that the creation of an additional window in the vestibular labyrinth is only justified when the stapes is not free to move in response to an acoustic stimulus.

Now I would like to digress for a moment on the subject of stapes fixation. If words mean anything, then surely "fixation" or "ankylosis" as applied to a joint mean immobility. But as we all know in otosclerosis the stapes is rarely immobile. Surely it would be nearer the truth to describe it as a stiff joint or a joint in which free movement is hindered or impeded. When the stapes is really fixed, little or no sound reaches the cochlea, and the signs are those of a perceptive deafness even though the nerve and end organ of hearing are intact. I mention this because I believe that in otosclerosis signs suggesting reduced cochlear function are often due to sound waves being prevented from reaching a normal organ of hearing, and not to changes in the organ of Corti or cochlear nerve. For some time now Littler and I have been trying to measure the impedance of the stapes in otosclerosis by nonacoustic methods; but I think that we can regard the deafness in otosclerosis as a good guide to the amount the stapes footplate is impeded by otosclerotic bone.

Now we come to mobilization of the stapes which has been so successfully developed by Rosen. I must confess that I have not read Minot's original work, and I still don't quite understand why it was abandoned and forgotten. However, I think that great credit is due to Rosen for having re-introduced it.

To date I have attempted 110 mobilizations with gratifying improvement in hearing in a quarter of the cases, and some improvement in another fifth. It is too early for me to say whether those improved will maintain their good hearing, and so far only one-tenth have lost their initial improvement. Out of 22 patients whose mobilization was unsuccessful and who were subsequently submitted to the formal fenestration operation, 21 ended with good hearing. Thus it seems that even if mobilization is unsuccessful it does not prejudice the success of a subsequent fenestration operation. Furthermore the fenestration operation is not made any more difficult by the previous failed mobilization.

Now for a few "Misadventures with the Stapes" in attempting mobilization. Pressure on the neck of the stapes can sometimes detach it from the crura. Either crus or both crura can be fractured, but the anterior is the more vulnerable of the two. For the actual mobilization I use the Zeiss microscope with 16 diameters of magnification. In this way one has an excellent, I might almost say exciting, view of all the structures and it is often possible to forestall a complete fracture of a crus by observing a slight crack so that a form of greenstick fracture is produced. This does no harm and is an indication that another form of manipulation is needed. Disruption of the incudostapedial joint does not appear to be as serious a mishap as at first sight it would seem. After getting the feel of the ossicular chain by pressing with a needle on the incudostapedial joint I go to the footplate using a needle at the anterior end which I try to mobilize by pushing it inwards. When this seems to be succeeding I then gently apply the mobilizer to the neck of the stapes to test its mobility. Sometimes it is necessary to apply pressure with the needle to the margins of the footplate a short way from the anterior end. Sometimes the needle catches on the anterior crus near its junction with the footplate resulting in a fracture. So far I have avoided breaking the footplate, and I have not as yet perforated the footplate as described by Rosen. One of my patients has had her hearing made a little worse by mobilization.

I have found that the operation is most likely to succeed when the hearing loss does not exceed 50 decibels.

The saying "To him that hath shall be given" applies to mobilization, because out of 19 patients with unilateral otosclerosis no less than 17 have had satisfactory improvement in hearing. Also in 11 patients in whom mobilization was successful in one ear, it was also successful in the second ear in eight.

I would now like to refer to destructive procedures upon the labyrinth for Ménière's disease via the oval window. Lempert described this approach in 1948, and Schuknecht also described it at the Triological meeting in 1956. Soon after starting mobilization in the autumn of 1955 I decided to take advantage of this approach to destroy the labyrinth. To do this, first of all the incus had to be removed and then the stapes had to be dislocated out of the oval

window. Then the accessible membranous labyrinth in the vestibule was removed. This was not easy because the utricle nearly always retracted upwards to hide behind the facial lip of the oval window. It could usually be brought down into the field by manipulation with a bent needle, or by applying a fine bent suction tube. Then it could be picked out with crocodile forceps whose jaws were small enough to go through the oval window. Then came the problem of how to close the oval window afterwards.

I found that if the stapes could be dislocated intact and turned to one side without severing its connection with stapedius, it was possible to replace the stapes in the oval window after removal of the membranous labyrinth. I have now carried out this procedure in 26 patients with unilateral Ménière's disease, and in all there has been a total loss of both cochlear and vestibular function. Doctor Shea has shown another way to close the oval window by cutting off the long process of the incus and the pushing the short process of the incus into the oval window.

It is of course a slighter procedure than the membranous labyrinthectomy via the lateral canal which I have practiced for nearly 20 years, but its main attraction is that it gives one the opportunity of working on the footplate of a healthy stapes and I believe that it will help us to a better appreciation of the problems of mobilizing a stapes impeded by otosclerotic bone. I would however utter a few words of warning, first of all, concerning the facial nerve. On five occasions I have found a dehiscence in the facial canal as it runs along the upper margin of the oval window so that the unprotected nerve sheath bulged into the oval window. In the first two of these cases the manipulations resulted in a transient facial paresis, but I hope that this will not happen again.

The other area to be careful of is the inner wall of the bony vestibule. As one views it through the oval window a faintly bluish area may be seen when using the microscope and this is a very thin plate of bone leading to the fundus of the internal auditory meatus. This can easily be cracked with pressure and a brisk and possibly prolonged flow of cerebrospinal fluid will result. This was the route taken by my chief Jenkins when he practiced translabyrinthine drainage for otogenic meningitis.

Finally I would like to say that in order to get the best out of the stapes, whether the operation be for deafness or for vertigo, it should always be viewed and manipulated under the highest magnification that can conveniently be used. In practice this is usually X 16.

You will be relieved to hear that at last I am coming to the end of my "Adventures with the Stapes," at any rate for the present. There is plenty more to do and whether we push it or rock it or pull on it, perforate it or remove it or by-pass it, the stapes deserves all the critical attention we are able to devote to it.

It is with pride and with pleasure that I can say that our knowledge of the stapes in health and in disease owes much to the inspired investigations of past and present members of the American Otological Society and I venture to predict that this Society of which I am so proud to be an Honorary Member will play a leading part in future advances in our knowledge of this fragile little bone.

149 Harley St.

XLII

STRUCTURE OF THE STRIA VASCULARIS AND THE SPIRAL PROMINENCE

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The structure of the stria vascularis has been the subject of many investigations. The specialized appearance of these epithelial cells enclosing an extensive capillary network, not duplicated elsewhere in the membranous labyrinth, has always aroused the interest of otohistologists. An excellent bibliography can be found in a recent publication by Nachlas and Lurie.[1] Some different functions have been ascribed to this region, but the most persistent has been that it is the site of endolymph formation.

Many studies have been undertaken in an effort to give some substance to this theory, some cytological in nature. Von Fieandt and Saxen[2] have made the most detailed observations on its cell structure. They described chromophil and chromophobe cells, the former showing plasma extensions which encircled the capillaries. They presented what they believed to be evidence for secretory activity in the changing position of the Golgi apparatus and related secretory granules.

Studies of organs active in the transfer of fluid have shown them to be characterized by a marked increase in the basal cell surface,[3] accomplished by either projections or infolding of the plasma membrane. Previous observations have shown some of the epithelial cells in the body of the utricle[4] to exhibit a similar modification. Engstrom, Sjostrand and Spoendlin[5] in a recent study with the electron microscope found many plasma extensions to be present in the cells of the stria vascularis of the guinea pig's cochlea. The purpose of the present studies was to determine whether any significant features

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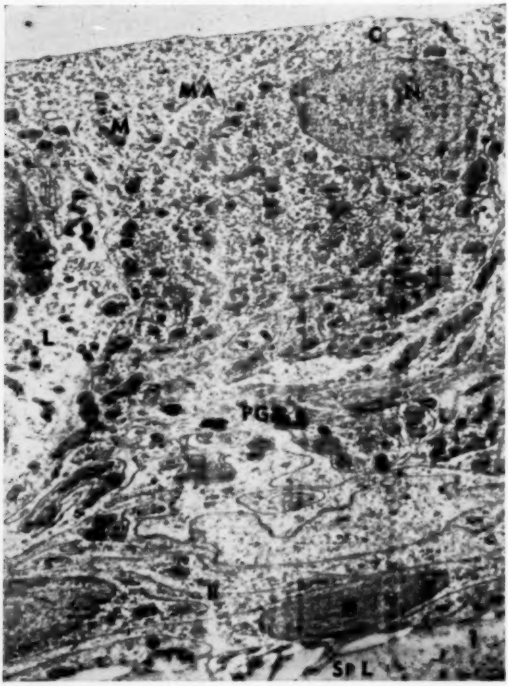


Fig. 1.—Electron micrograph showing the entire thickness of the stria vascularis. A marginal cell (MA) faces the endolymph. The cytoplasm is filled with membrane structures, mitochondria (M) and a granular material. Some large, round cisternae (C) are visible near the surface. The basal part of the cell is not well defined because of the manner in which the extensions from it interlock with those from other cells. Part of a light (L) cell is visible at the left. The flat basal cells (B) border the spiral ligament (SpL). Pigment granules (PG); Nucleus (N). x 7,200.

were present in the stria vascularis and spiral prominence and to compare these findings with those already made on organs active in fluid transport.

METHODS

The ears of seven guinea pigs were fixed by perfusing the cochleas of the anesthetized animals with 1% osmic acid buffered with sodium veronal[6] or according to Dalton,[7] to a pH of 7.2 to 7.7. The animals were decapitated and the excised ears immersed in the fixative for a total fixation period of one hour. Pieces of the spiral ligament, with the stria vascularis attached, were dissected free from the cochlea, dehydrated in ethanol and embedded in a methacrylate mixture (1 part methyl plus 10 parts butyl methacrylate). Thin sections were cut by means of a Porter-Blum microtome and viewed in a RCA electron microscope, model EMU2E. Photographs were made at original magnifications of 1900x to 10,000x. Micrographs were printed at enlargements of 4x or better.

OBSERVATIONS

Stria Vascularis. The stria vascularis is a compact band of epithelial cells, which with the cells of the spiral prominence forms the lateral wall of the cochlear duct. A double layer of thin, flat cells (the basal cells) separates it from the spiral ligament. The capillary network is found in the epithelial layer. It is enclosed by the marginal cells, which face the endolymph, and other cells of less dense cytoplasmic content.

Figure 1 shows a section through the entire thickness of the stria vascularis. A marginal cell, which is the predominant type, occupies the upper half of the micrograph. It appears rectangular in shape, but it is doubtful if the section is made through the entire cell. The isolated cell segments with the large mitochondria, below the pigment granules, are probably sections of the cell's extensions. The free surface which contacts the endolymph is straight and smooth. Other micrographs show a few protuberances, but no microvilli. The lateral plasma membrane close to the free surface shows condensations of osmiophilic material between adjoining cells. These are similar to the system of terminal bars found between the epithelial cells in

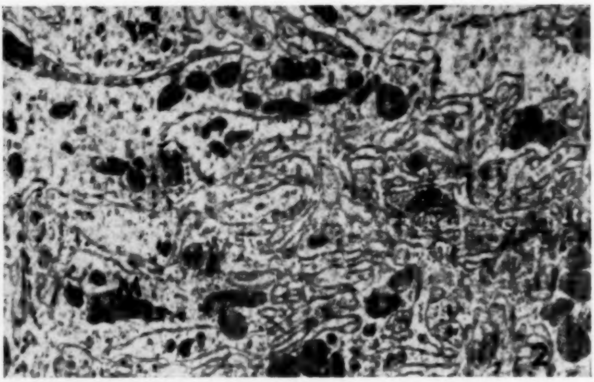


Fig. 2.—Electron micrograph showing the complex nature of the basal surface of a marginal cell. The membranes visible represent extensions and infoldings of the basal plasma membrane. Mitochondria (M). x 9,500.

others parts of membranous labyrinth. The plasma membrane of adjacent cells is adherent for about half the length of the cell. Then it begins to dip inward forming deep furrows, or long finger-like projections. This pattern becomes more elaborate toward the base of the cell, and the entire basal cell membrane is composed of infoldings and projections.

The infolded basal surface is more clearly illustrated in other photomicrographs, as Figure 2. No regularity is obvious. Apparently, the plasma membrane is infolded upon itself and also forms projections which interdigitate with those from other cells. The interdigitation seems to be more prominent near the spiral ligament where the marginal cell processes interlock with those from the lighter cells (Figs. 1 and 3). Around the blood vessels, the pattern appears to be one primarily of infolding (Figs. 4 and 5) with processes from the light cells less evident. Figures 4 and 5 show the extensive infolding present about the capillaries. The membranes are both perpendicular and parallel to the endothelial cell membrane in the same section, giving evidence that long projections may pass along the endothelium for a short distance. Figure 5 shows a marginal cell as it abuts upon

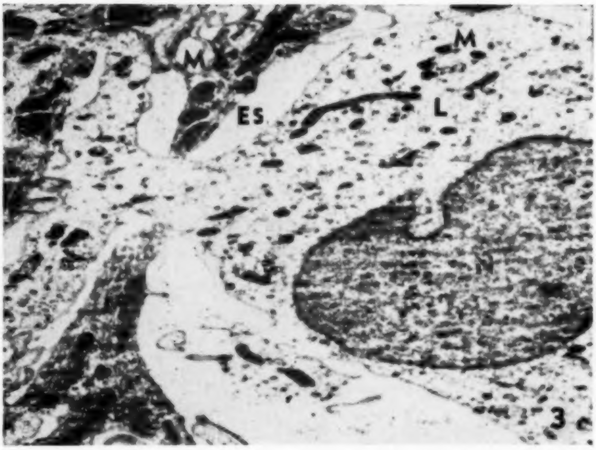


Fig. 3.—Electron micrograph showing one of the light cells. The cytoplasm is less osmiophilic than that of the two marginal cell processes (MA) at the left. The mitochondria (M) are smaller. Nucleus (N); Extracellular space (ES). x 11,400.

the capillary endothelium. The two cell walls are separated by a thick basement membrane. The spaces between the infolded plasma membrane of the marginal cell are narrow, relatively constant and of low density. Micrographs from some ears which appear to have been unusually hydrated, have shown the membranes to be widely separated and a granular material similar to that of, and apparently continuous with the basement membrane to extend for some distance up into the open spaces.

The cytoplasm of the marginal cells is dense (Figs. 1 and 6). It contains large mitochondria, a heavy concentration of granules and many membrane structures. The latter vary in size and shape. Small round vesicles and globules, or slightly elongated sacs predominate. Most show attached granules. Several membrane groups which are apparently Golgi apparatus are usually visible. Some larger cisternae containing a sparse granular material are often found in the upper part of the cell. In the basal part of the cell, or where the infoldings

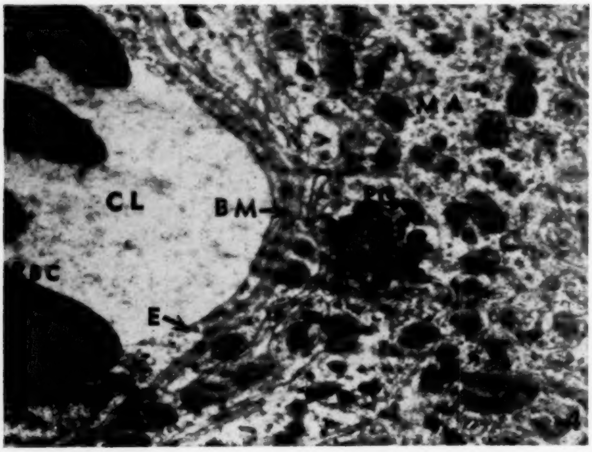


Fig. 4.—Electron micrograph showing edge of a capillary and closely applied marginal cell (MA). A thick basement membrane (BM) separates the endothelial cell (E) from the marginal cell processes. Capillary lumen (CL); Pigment granules (PG); Erythrocytes (RBC). x 14,250.

are numerous, membranes of a different character are found. Figure 7 is a section made from an area in the vicinity of a blood vessel. Rows of small round vesicles without granules are visible among the mitochondria. These beaded arrangements are sometimes found in other cells in limited numbers and seem to be vesicles pinched off from infolded plasma membranes. Figure 8 illustrates such a phenomenon in an external spiral sulcus cell. Examples can also be found in the marginal cells which show a comparable continuity with the plasma membrane, but are more difficult to demonstrate, because of the complex character of the epithelium. The abundance of the beaded vesicles in the basal part of the cell is consistent with the viewpoint that they originate from infolded cell membranes.

The mitochondria are scattered throughout the marginal cells, but are found in greater numbers in the basal portion. They are large and vary in shape from spheres to rods. They are particularly long in some of the projections, where they may occupy almost the

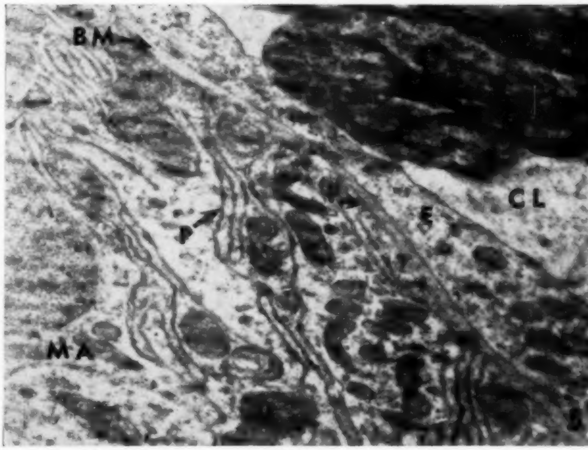


Fig. 5.—Electron micrograph showing relationship of the infolded plasma membrane (P) of the marginal cell (MA) to the capillary endothelium (E). An erythrocyte is at the upper right. Capillary lumen (CL); basement membrane (BM); mitochondria (M). x 22,800.

entire cytoplasmic area. Their internal structure is consistent with that described for mitochondria in other cells.

The nucleus seen in Figure 1 is oval in shape, with an indentation. It is filled with a granular material, evenly distributed. The nucleus is always close to the free surface, and usually displaced to one side.

The lighter cells (Fig. 3) found between the marginal and basal cells, are fewer in number and rarely reach the free surface. The cytoplasm contains only a scattering of ergastoplasm. The aggregates of Golgi apparatus can readily be distinguished in these cells. The mitochondria are mostly rod-shaped. One long filamentous mitochondria is visible in the cell in Figure 3. The nucleus is often indented and occupies a position toward the base of the cell. These cells likewise exhibit projections of the plasma membrane. Figure 3 shows a large projection interlocking with others from a marginal cell. The contrast in cytoplasmic density between the two cells is well marked. The processes formed by these cells are usually large

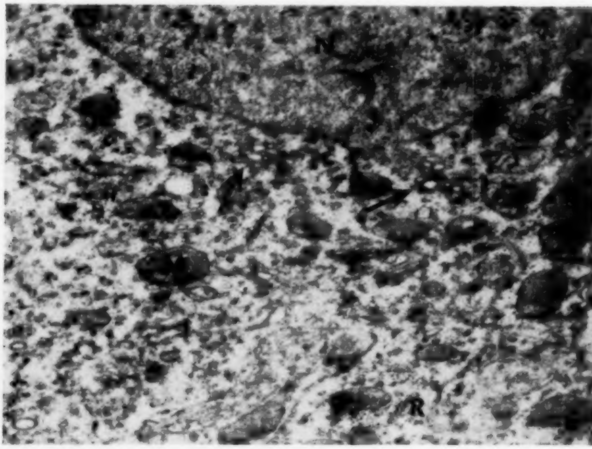


Fig. 6.—Electron micrograph showing the cytoplasm of a marginal cell at higher magnification. The ergastoplasmic sacs (R) and other membranes of smooth contour are scattered in abundance. Two membrane groups (arrows) are probably Golgi Apparatus. Nucleus (N); Mitochondria (M). x 22,500.

and thick. The elaborate infolding found in the marginal cells does not seem to be present.

The light cells often contain pigment granules. Most of the pigment present in the stria vascularis is found in these cells. Only an occasional granule has been observed in the marginal cells.

The basal cell layer (Fig. 1) separates the stria vascularis from the spiral ligament. It is one to several cells in thickness. The cells have flat, thin plates of cytoplasm spreading out from the oval nucleus, which is centrally located. The cytoplasm shows the usual organelles, i.e., endoplasmic reticulum and mitochondria, and a sparse granular material.

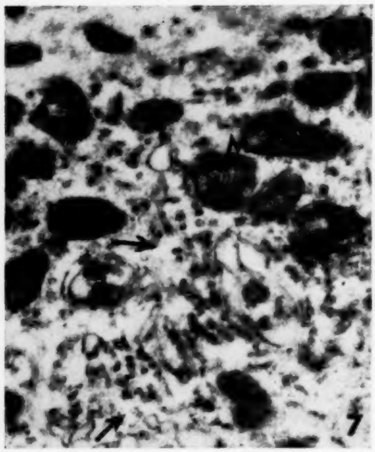
Spiral Prominence. Figure 9 shows a section of the spiral prominence. It is covered by one layer of osmiophilic cells. These cells are arranged in a single row extending from the stria vascularis to the

external spiral sulcus, where there is an abrupt transition in cell structure. They rest on a basement membrane which separates them from the spiral ligament below. The cells from the basal layer of the stria vascularis underlie the first few cells at the upper limits, and the epithelial extensions from the external spiral sulcus may pass under the lowermost cell. For the most part, Figure 9 is typical. A space, loosely filled in with fibrous material and scattered cell processes lies between the spiral prominence epithelium and the cells of the spiral ligament. These latter are packed more tightly together in the prominence than elsewhere in the spiral ligament.

The plasma membrane of the epithelial cells shows microvilli on the free surface. Adjacent cells are tightly joined as evidenced by the terminal bars near the free surface. Basal to the terminal bars, th plasma membranes separate and form innumerable finger-like projections. These loosely interdigitate with others from the same or adjoining cells. Definite extracellular spaces are visible about the projections, at times giving the entire cellular layer a lacy appearance. Figure 10 shows the projections at higher magnification. The larger open spaces are usually found bordering the spiral ligament. Many of the processes lie parallel to the basement membrane, creating the impression that the fluid spaces may be intracellular. A continuous plasma membrane limiting the processes and the many points where the spaces are separated from the spiral ligament only by a basement membrane give clear evidence that these are extracellular spaces.

The cytoplasm is dense (Fig. 10) and difficult to analyze. The membranes present are mostly in the form of small round or oval vesicles. Some cisternae which may contain a sparse granular material are usually found near the surface. It cannot be determined whether these are actually intracellular vacuoles, or the ends of the extracellular spaces. Some of the micrographs show considerable cytoplasmic vacuolization, but this may be fixation artefact. The round or rod-shaped mitochondria are scattered throughout the cell and sometimes are seen in the projections. The remainder of the cytoplasm is filled with a fine granular material, only slightly less dense than the nuclear material. The nucleus is usually lobulated. It occupies a large part of the cell.

Blood Vessels. Figures 4 and 5 show sections of capillaries embedded in the stria vascularis. The nucleus of the endothelial cell



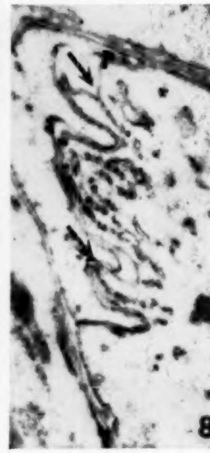


Fig. 7.—Electron micrograph of basal cytoplasm of a marginal cell showing typical rows of small round vesicles (arrows). Mitochondria (M). x 27,000.

Fig. 8.—Electron micrograph of an external spiral sulcus cell showing beaded cytoplasmic membranes similar to those in Fig. 7. Note their close relationship to infolds (arrows) in the plasma membrane (P). x 18,000.

is oval in shape and creates a bulge in the vessel wall. The remainder of the cell is formed by a thin cytoplasmic layer which varies in thickness, but is apparently continuous around the capillary. A thick basement membrane completely surrounds the endothelial cell wall. A second cell layer is occasionally seen. Whether this is part of the vessel wall or one of the strial cell processes extending parallel to the endothelium is uncertain. The latter seems most likely.

The cells of the stria vascularis completely surround the capillaries. A process from one of the light cells, sometimes with pigment, may cover a sizeable area of the endothelium but the infolded surfaces of the marginal cells predominate. Figure 5 shows that while the plasma membrane of the marginal cell is infolded in a complicated manner, the surface it presents is parallel to that of the endothelial cell. Some micrographs have shown the granular material of the

basement membrane to extend back into the spaces sometimes present between the cellular projections.

The vessel of the spiral prominence is not found in the epithelium, but in the spiral ligament. The prominence itself is actually a protuberance of the spiral ligament cells with some epithelial projections from the external spiral sulcus. This is covered by the osmic-dense prominence cells. The vessel of the prominence may have an adventitial cell superimposed about the endothelium. The basement membrane is distinct, but thinner than that surrounding the capillaries of the stria vascularis. The vessel is loosely suspended among the cells of the spiral ligament. Processes from these cells and some loose fibrous material separate it from the basement membrane which supports the epithelium of the prominence.

COMMENT

These observations and those made recently by Engstrom, Sjostrand and Spoendlin[5] have confirmed the earlier observations of von Fieandt and Saxen.[2] The electron microscope can only add the cytological details to the description of the stria vascularis already given in the beautiful studies of von Fieandt and Saxen. Their "chromatophil cells" are identifiable with the marginal cells. The high ergastoplasmic content apparently increases their stainability. The extensive infolding of the basal plasma membrane was not visible by ordinary light microscopy, but the larger projections were recognized by von Fieandt and Saxen. The "fibrillar striations" were described in the same regions where the electron microscope has revealed the filamentous mitochondria and beaded vesicles.

The cells which cover the spiral prominence are smaller and less well defined by ordinary methods. Von Fieandt and Saxen identified some short processes and apparently believed them to be similar to the cells of the stria vascularis. The electron microscope has revealed cytological differences between the epithelium of the two regions although, in some respects, the basic pattern is similar. The spiral prominence cells have a more osmiophilic cytoplasm with a heavy concentration of granules. In contrast, the marginal cell cytoplasm has a high ergastoplasmic content as well as other membrane structures. The latter do not show microvilli. The prominence epithelium more closely resembles the cells found in the body of the utricle[4]

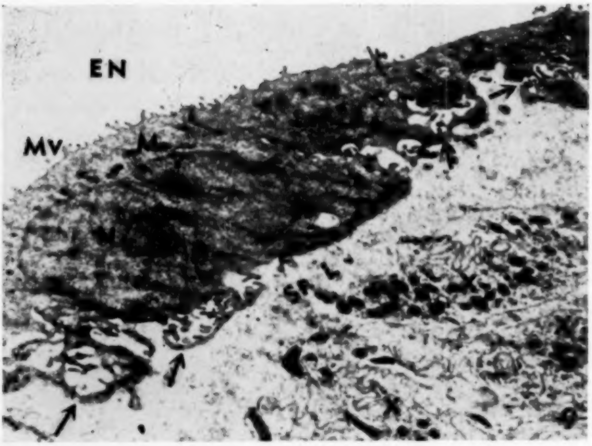


Fig. 9.—Electron micrograph showing the spiral prominence. The epithelial cells facing the endolymph (EN) are highly osmiophilic. The basal plasma membrane forms many short, thick projections (arrows) which extend into large extracellular spaces. Three spiral ligament cells (X) are visible at the lower right. Microvilli (MV); Nucleus (N); Mitochondria (M); Spiral ligament (SpL). x 8,550.

and the ampullae[8] where cellular processes project into large extracellular spaces separated from the perilymph by a basement membrane. The cochlear cells lack the closely related pigment cells which accompany those in the vestibule. The cells of all these regions, in vestibule and cochlea, however, show the common characteristic of a considerable increase of basal plasma membrane accomplished by means of cellular projections. Those of the marginal cells are crowded together so that they seem to be infoldings with a negligible interposed space, whereas over the spiral prominence they project singly into large intracellular spaces and appear as finger-like projections. In instances where the stria vascularis has been unusually hydrated and the processes well separated, the resemblance between the two patterns is more evident.

An electron microscopic study of organs with cells that show intensive infolding of the basal plasma membrane has recently been

made by Pease.[3] He found this type of structure in the kidney tubules, serous alveoli and secretory duct of the submaxillary gland, the choroid plexus and ciliary body. To these can now be added the stria vascularis. The renal glomerulus,[9] placenta and mammary gland[10] show modifications which more closely resemble the projections surrounded by fluid spaces that are found in the spiral prominence and vestibule. All of these organs studied are epithelial structures noted for their fluid transport, and it seems reasonable to assume the same process is occurring in cells of similar structure in the membranous labyrinth.

The beaded vesicles in the basal portion of the marginal cells give an even firmer basis for this assumption. Among the organs he studied, Pease[3] found these membrane arrangements only in the ciliary body. Palade[11] observed them in macrophages and suggested they may be a route for pinocytosis and phagocytosis. Their presence in such quantities close to the infolded membranes of the marginal cells suggest they play an important part in the physiology of these cells, one which may well be related to the intake of fluid.

The evidence lending support to the theory that the stria vascularis is the site of endolymph formation is mostly of an indirect nature. The presence of a rich capillary network surrounded by deeply staining cells is suggestive of secretory function. Various experimental studies, among which Guild's[12] and Altman and Waltner's[13] are probably the best known, have shown that while injected granules may accumulate elsewhere in the cochlear duct, they are seldom found in the stria vascularis. Cellular debris which is dislodged from the basilar membrane by acoustic trauma is rarely seen close to the stria vascularis.[14] The structural evidence presented here is compatible with the theory that the stria vascularis is engaged in fluid passage. The weight of the sum of all these pieces of evidence is such that there can be little doubt but that the stria vascularis and spiral prominence do participate in the production of endolymph.

The stria vascularis, however, is a unique structure in the entire membranous labyrinth. It seems certain that endolymph must be formed and absorbed in the vestibule as well as in the cochlea. Yet, it is the cells covering the spiral prominence that are duplicated in the vestibule, not the stria vascularis. It may be that the absence of

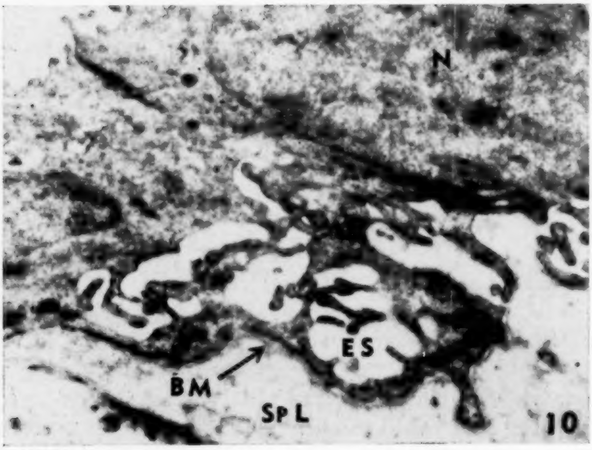


Fig. 10.—Electron micrograph of area at lower left in Figure 9 at higher magnification. The cytoplasm is almost as osmiophilic as the nuclear material (N). A basement membrane (BM) separates the epithelium from the spiral ligament (SpL). Extracellular space (ES). x 18,000.

fluid accumulation in the latter is indicative of the speed of the process, and transfer is only more rapid in the stria vascularis. The cytological specialization of the marginal cells suggests some additional process and the question arises as to whether it may not be also the site of some activity specific to the cochlea. Recent experiments at Central Institute for the Deaf in St. Louis, with the co-operation of the Department of Otolaryngology of the University of Chicago,[15] have given evidence that the stria vascularis may also be responsible for the production of the large positive DC potential found only in the cochlear duct.

SUMMARY

Observations with the electron microscope have revealed the cytological details of the cells of the stria vascularis and spiral prominence. The major cells of the stria vascularis are the marginal cells, which face the endolymph. The cytoplasm of these contains many

membrane structures, granular material and large mitochondria. The basal plasma membrane is greatly increased by infolding and projections, particularly about the capillaries. Some other cells, the light cells, which rarely reach the endolymph surface have a less dense cytoplasm. The epithelial cells which cover the spiral prominence are more osmiophilic than those of the stria vascularis. The basal plasma membrane shows many processes which project into extracellular spaces, separated from the spiral ligament by basement membrane.

EUCLID AVE. AND KINGSHIGHWAY

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REFERENCES

1. Nachlas, N. E., and Lurie, M. H.: The Stria Vascularis: Review and Observations. Laryngoscope 61:989-1003, 1951.

2. von Fieandt, H., and Saxen, A.: Beitrage zur Histologie der Stria Vascularis und der Prominentia spiralis bei Saugern (Hund und Mensch). Ztschr. f. Anat. u Entschlngegesch. 106:425-446, 1936-37.

3. Pease, D. C.: Infolded Basal Plasma Membranes Found in Epithelia Noted for Their Water Transport. J. Biophysic. and Biochem. Cytol. 2 Suppl.: 203-208, 1956.

4. Smith, C. A.: Microscopic Structure of the Utricle. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 65:450-470, 1956.

5. Engstrom, H., Sjostrand, F. S., and Spoendlin, H.: Feinstruktur der Stria vascularis beim Meerschweinchen. Pract. Oto-rhin-laryng. 17:69-79, 1955.

6. Palade, G. E.: A Study of Fixation for Electron Microscopy. J. Exp. Med. 95:285-298, 1952.

7. Dalton, A. J.: A Chrome-osmium Fixative for Electron Microscopy. Anat. Rec. 121:281, 1955.

8. Smith, C. A.: Unpublished data.

9. Pease, D. C.: Electron Microscopy of the Vascular Bed of the Kidney Cortex. Anat. Rec. 121:701-722, 1955.

10. Dempsey, E. W., and Wislocki, G. B.: Electron Microscopic Observations on the Placenta of the Cat. J. Biophysic. and Biochem. Cytol. 2:743-754, 1956.

11. Palade, G. E.: The Endoplasmic Reticulum. J. Biophysic. and Biochem. Cytol. 2 Suppl.: 85-97, 1956.

12. Guild, S.: Circulation of the Endolymph. Am. J. Anat. 39:57-81, 1927.

13. Altmann, F., and Waltner, J. G.: Further Investigations on the Physiology of the Labyrinthine Fluids. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 59:657-686, 1950.

14. Smith, C. A., and Covell, W. P.: Unpublished data.

15. Davis, H., Deatherage, B. H., Fernandez, C., Kimura, R., Rosenblut, B., and Smith, C. A.: The Modification of Cochlear Potentials Produced by Streptomycin Poisoning and by Extensive Venous Obstruction. In press.

XLIII

EXPERIMENTAL OBSTRUCTION OF VENOUS DRAINAGE AND ARTERIAL SUPPLY OF THE INNER EAR

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Vascular accidents in the labyrinth may account for sudden loss of function in a manner similar to that which occurs in the central nervous system. The mammalian labyrinth is almost completely dependent on its vascular system for a continual supply of oxygen, and other metabolites. Within seconds after respiratory oxygen is replaced by nitrogen or after general blood flow is impaired functional changes are demonstrable. The vascular system of the labyrinth plays an important but obscure role in health and disease. This has been difficult to assess in the clinic and in the laboratory. The post-mortem degeneration in human material precludes an evaluation of the finer histological changes in the labyrinth. Correlation of histologic findings in the labyrinth vessels with other histologic changes and with clinical symptoms is particularly difficult. In animals differences in fixation and similar technical details have caused some confusion in interpretation. Furthermore experimental means for producing discrete vascular lesions of the labyrinth have been limited.

In our laboratory a number of vascular lesions have been produced experimentally. This report is concerned with the histological changes following two types of vascular lesions, one sudden extensive venous obstruction, the other sudden arterial obstruction of the labyrinth. Preceding these investigations a number of vascular injections with Prussian Blue and with lead chromate were made to study the distribution of these blood vessels and their surgical accessibility.

From the Division of Otolaryngology of the University of Chicago. This work was aided in part by funds from U.S. Public Health Grant B269 (C4) and Douglas Smith Foundation for Medical Research of the University of Chicago.

Fig. 1.—Section through cochlea showing the relation of the inferior cochlear vein (V C C) to the bulla cavity, posterior ampulla (PA), cochlear aqueduct (CA) and the posterior spiral vein (V S P).

Investigators agree that all the arterial vessels to the labyrinth branching in a variety of ways enter along with the VIII nerve trunks through the internal acoustic meatus. There is however some disagreement about the venous drainage. In the human Siebenmann[1] describes three veins, one adjacent to the cochlear aqueduct, another along the endolymphatic sac, and one through the internal meatus. Nabeya[2] did not find this last named vessel and called attention to differences in venous drainage among species. The inferior cochlear vein accounts for a large part of the venous drainage of the cochlea in man, and according to Nabeya for a large part of the venous outflow

of the labyrinth in the guinea pig. This experimental animal was chosen for the vascular study particularly because the vein is surgically accessible and can be interrupted without damage to the cochlear aqueduct or the cochlea (Fig. 1). In this animal as in man the inferior cochlear vein courses in a minute bony channel from the basal coil to the inferior petrosal sinus adjacent to, but entirely separate from, the bony channel of the cochlear aqueduct.

VENOUS OBSTRUCTION

In earlier experiments the vein was interrupted some distance from the basal coil and produced very minimal changes. Subsequent examination of injected material and of Nabeya's account indicated that a branch to the internal meatus is rather constant and is given off from the inferior cochlear vein very close to the basal coil. In addition we noted one or two small tributaries from the middle ear joining the inferior cochlear vein. Interruption of the vein and its tributaries at the base of the cochlea rather consistently caused a change in cochlear blood flow as observed by us in the spiral ligament and in the stria vascularis.[3] Dilation of vessels and slowing of blood flow up to 90 per cent of the normal rate could be produced. Complete cessation of flow in the stria capillary often followed such a lesion. A series of animals were then operated on under sterile conditions and sacrificed at intervals so that the progressive changes could be assessed. Evidence for profound functional disturbance was noted in the vestibular signs and in the electrical activity of the cochlea. The resultant nystagmus, head and body torsion and circling movements resembled that seen in acute labyrinth destruction. Recovery usually occurred in a few days, but in some animals prolonged head torsion was noted.

The animals were sacrificed at intervals by intravital perfusion with a small volume of warm normal saline followed by Heidenhain-Susa solution at 38° and under 30″ of water pressure. Then the entire skull base containing both labyrinths and the brain stem between them was removed in block and after decalcification, etc., sectioned at 18 micra. The method of fixation and the fixative used are particularly good for preventing distortion of the end organ and ganglion cells. The method of preserving both labyrinths on the same slide permits a careful evaluation of histological changes on the affected ear by comparing it with the normal ear. At time of sacrificing the animal, some of the cochleas were fenestrated for evi-

dence of abnormal blood flow. Absence of the striae capillaries with preservation of arteriovenous arcades was striking in some cases of prolonged venous obstruction.

Within 24 hours after venous obstruction loss of outer hair cells was clearly evident. In more chronic animals, changes were seen throughout the cochlear duct but the basal turn was more severely involved. The inner hair cells were more resistant to change than the outer hair cells. Small, scattered areas of degeneration were found among the inner hair cells after two days.

Ganglion cells were reduced in number two weeks after obstruction and this was very extensive in the five months old lesion—though not total even when the hair cells were degenerated throughout the cochlea. The stria was particularly susceptible often showing the first signs of damage. Dilation of strial capillaries and edema of the epithelium were noted one hour after venous obstruction. Within a few hours hemorrhage into the epithelium and into the scala media was noted. Disintegration of the epithelial layer of the stria soon followed in some. Various changes were noted at one week; there was hypertrophic epithelium with highly dilated capillaries; partially denuded epithelium in the region close to the spiral prominence, flat atrophic epithelium with marked reduction in the number of capillaries, and finally simple cuboidal epithelium without capillaries.

In the spiral ligament, the cells in the region adjacent to the cells of Claudius and the external sulcus cells showed the first change—pyknosis in six hours. By two weeks this area became acellular. This spread behind the stria in the more severe lesions.

Hemorrhage from the strial capillaries was seen in the cochlear duct up to seven days after obstruction. Cellular fragments and pigment granules released from the degenerated stria were present throughout the experimental period of six months. Neither the blood cells or the cellular debris in the cochlear duct showed indication of movement toward the saccule. Bleeding into the perilymphatic space was commonly seen around the collecting venule. By the sixth day this blood aggregated in the upper turns of the cochlea where it remained for some time. At the end of five months all traces of blood had disappeared. Many phagocytes were found in the area of hemorrhage.

Over 80 per cent of the animals showed vestibular disturbances and histological changes in the vestibular labyrinth. The saccule was affected as frequently as the organ of Corti. The sensory cells were the most vulnerable. The first signs of damage were noted six hours after obstruction. Pyknosis and fragmentation of nuclei were followed by edema and separation of the hair cell layer from the supporting cells with displacement of the otolithic membrane. This continued to complete degeneration of the hair cell leaving only a thin layer of supporting cells. Hemorrhage in the saccule was less frequent than in the cochlear duct. No dilation of the saccule was observed. The sensory cells of the utricle were also vulnerable and underwent changes similar to those seen in the saccular macula. In addition there was considerable activity of the pigment cells normally dispersed along the walls of the utricle. Pseudopodial and spherical shapes as well as pigment granules of disintegrated cells were seen. Hemorrhage was rarely seen within the utricle but was often severe in the perilymphatic meshwork around the utricle. The meshwork retained the cells so that the rest of the vestibule was clear. Cellular fragments and blood cells within the utricle did not appear to migrate towards the utriculo-endolymphatic valve. The sensory cells of the cristae were as commonly involved as those of the utricle but in severe lesions the supporting cells also were affected. Pyknosis and fragmentation of sensory cell nuclei were followed by edema and separation of sensory cells from the supporting cells and finally by disintegration. Partial degeneration of the nerve fibers was evident but Scarpa's ganglion cells appeared normal. Extensive hemorrhage was also seen in the perilymphatic meshwork around the membranous canals. In this area fibrosis and even ossification was seen in the chronic states.

The epithelial cells of the endolymphatic sac showed no change but the contents of the sac appeared to stain more deeply with eosin and have more phagocytes than on the normal side. There was no apparent change in the size of the sac.

The findings in this experiment indicate the severe damage to the cochlear and vestibular sensory elements, the hemorrhage in the perilymphatic and endolymphatic spaces, etc., that may follow sudden venous obstruction of the labyrinth. The supplementary venous channels along the endolymphatic duct and into the internal meatus

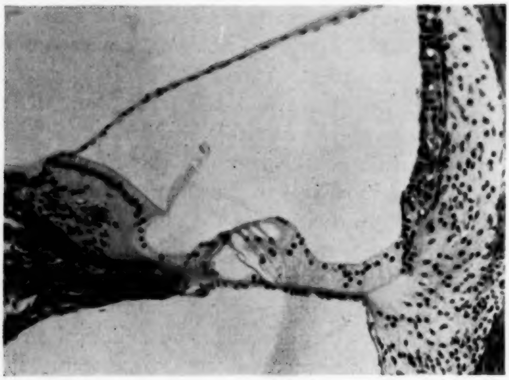

Fig. 2.—Normal cochlear duct. Compare with 3.

may protect the labyrinth particularly if there is a slowly developing obstruction in the inferior cochlear vein. On the other hand it is likely that blood flow in this vein in the human ear as it courses along the cochlear aqueduct in its minute bony channel may stop suddenly. Since it carries a large part of the venous blood from the cochlea auditory symptoms could be reasonably expected. It is interesting that dilation of the cochlear duct does not follow this vascular lesion. It is further of interest that the distribution of the cellular elements in the endolymphatic spaces as seen in the various lesions for periods up to six months does not indicate a systematic movement of this fluid towards the endolymphatic sac. The reactive fibrosis following hemorrhage about the membranous canals is noteworthy, particularly in contrast to its absence in the cochlea. Hemorrhage into the labyrinth in leukemia is well documented and offers one clinical and pathologic analogy to this experimental lesion.

It appeared logical to compare the effect of arterial obstruction on the labyrinth with that seen following venous obstruction. For this purpose a surgical approach through the bulla was worked out. The surgical field was adjacent to but independent from the inferior cochlear vein or cochlear aqueduct. With a fine dental drill the region of the internal meatus was approached medial to the basal turn.

the dura, including the inferior petrosal sinus, was elevated medially, then the dura was slit over the region where the anterior inferior cerebellar artery sends its branches to the internal meatus. At this point about 3 mm from the cochlear nerve the artery was electrically coagulated, after gently lifting it away with coagulating suction tip from the underlying brain stem. Lesions described in this report include intervals from 30 minutes to two weeks. Anatomical variations in the arrangement of blood vessels at the exposed site made for differences in the results obtained. The acute vestibular disturbances manifested by this disruption of the labyrinth circulation were similar to those seen following venous obstruction but probably more severe. The effectiveness of the operation was checked in preliminary studies and at times in the chronic preparations by direct observation of the circulation. In the acute experiments complete cessation of flow in the stria and spiral ligament was noted in a successful operation. In the chronic lesion, actual disappearance of blood vessels in these areas was observed.

The material was prepared as in the case of venous obstruction so that the operated side could be compared with the unoperated side on the same slide. In general interruption of the arterial supply produced more rapid and more profound changes in the labyrinth (Figs. 2 and 3). Within 30 minutes abnormalities of the inner hair cells could be clearly seen. In a few hours both the inner hair cell and the inner pillar begin to disintegrate. This was soon followed by involvement of the entire end organ. At the same time the outer sulcus cells, the inner sulcus cells, the cells of the spiral prominence as well as those of the spiral ligament and limbus spiralis also showed clearly defined changes. The stria vascularis showed striking change. These vessels remained filled with blood and showed no evidence of hemorrhage. The entire stria became detached in four hours. By the end of 17 hours the entire end organ and stria were fragments in the cochlear duct. As time progressed the spiral ligament and limbus spiralis became acellular. The basilar membrane was denuded. The cells of Reissner's membrane disappeared leaving a thin intact acellular basement membrane.

The changes in the spiral ganglion cells were seen in the early lesions. Reduction of Nissle substance and pyknosis was evident in a few hours. This continued on to the marked shrinkage or disap-

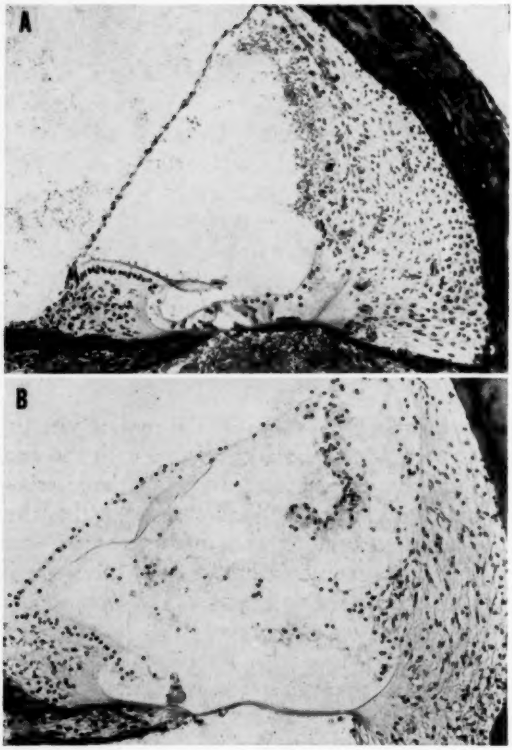

Fig. 3.—A. Changes in the cochlear duct one day after venous obstruction showing fragmentation of the stria and hemorrhage in the scala tympani with early changes in the end organ and spiral ligament.

B. Changes in the cochlear duct 17 hours after arterial obstruction showing dissolution of the organ of Corti and stria vascularis with marked cellular changes in the limbus, spiral ligament and Reissner's membrane.

pearance of ganglion cells in eight to 14 days. Changes in the nerve fibers in the modiolus were first noted in the Schwan cells and were later followed by the formation of myelin droplets. Rasmussen's bundle also showed degeneration. The vessels in the modiolus and in the rest of the cochlea were filled with blood or narrowed.

The vestibular labyrinth was less often affected but all changes appeared from edema of the maculae and its separation from the basement membrane to complete disintegration. The lesions were more severe than in venous obstruction. Perilymphatic hemorrhage was strikingly absent. The manner of branching of the anterior inferior cerebellar artery at the point of surgical interruption indicated that the anterior vestibular branch supplying the utricle and the superior and horizontal ampulla might escape injury while the cochlear trunk was blocked. Again the cells of the endolymphatic sac showed no change even in the face of the most severe degeneration.

By the end of a week fibrosis of the perilymphatic spaces had begun, particularly in areas of the cochlea near the round window. This preliminary report on arterial obstruction deals with changes occurring within the first two weeks. A more detailed report, including lesions up to six months' duration, will appear later.

The extreme changes produced by this vascular lesion resembles that previously described by Knick[4] incidental to VIII nerve section and six days after operation. A systematic picture of the progressive changes from hour to hour after arterial obstruction was not clearly developed by previous investigators. Later investigations[5,6] were directed to identification of the electrical phenomena of the cochlea and indicated the importance of the blood supply. The careful animal experiments on post-mortem degeneration in the labyrinth are of particular interest. For example, the clearly illustrated findings of Wittmack and Laurowitz[7] in 1912 and recently by Fernandez[8] are not unlike that seen soon after these vascular lesions.

The post-mortem changes seen in human material makes it particularly difficult if not impossible to evaluate the pre-mortem adequacy of the labyrinth vascular system. Only the most advanced lesions would be recognizable.

Again as with venous obstruction, it is noteworthy that change in the size of the endolymphatic space did not occur. Neither collapse or dilation of the cochlear duct was noted. Unlike the condition of venous obstruction, arterial obstruction did not produce hemorrhage into the perilymphatic and endolymphatic spaces. Of interest also is the swelling of the tectorial membrane and cupular substance as compared to venous obstruction. The changes in the nerve fibers are distinctly more severe and earlier than those following venous obstruction. Spiral ganglion cell changes are also more rapidly produced. The arterial occlusion produced in these animals is not complicated by simultaneous surgical damage to the nerve trunk. The histological changes reported are directly the result of arterial obstruction. The associated immediate changes in the brain stem nuclei, etc., seen in our material are also the result of interrupted circulation.

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REFERENCES

1. Siebenmann, F.: Blood Vessels in the Human Ear. J. F. Bergmann, Wiesbaden, 1894.

2. Nabeya, D.: A Study in the Comparative Anatomy of the Blood Vascular System of the Internal Ear in Mammalia and Homo (Japanese). Acta Scholae Medicinalis 6:1-132, 1923.

3. Perlman, H. B., and Kimura, R. S.: Observations of the Living Blood Vessels of the Cochlea. Annals of Otology, Rhinology and Laryngology 64:1176, 1955.

4. Knick, A.: Pathologische Histologie des Ohrlabyrinths nach Durchschneidung des Naevus acusticus. Ztschr. f. Ohrenheilkunde 65:342-378, 1912.

5. Hallpike, C. S., and Rawdon-Smith, A. F.: The "Wever and Bray Phenomenon." A Study of the Electrical Response in the Cochlea with Especial Reference to Its Origin. Jour. Physiol. 81:395-408, 1934.

6. Wever, E. G., and Neff, W. D.: A Further Study of the Effects of Partial Section of the Auditory Nerve. Jour. Comp. and Physiol. Psychol. 40:217-226, 1947.

7. Wittmaack, K., and Laurowitsch, Z.: Über artefizielle, postmortale und agonale. Beeinflussung der histologischen Befunde im membranösen Labyrinthe Zeitschrift für Ohrenheilkunde 65:157-189, 1912.

8. Fernandez, C.: Post Mortem Changes in the Vestibular and Cochlear Receptors. Air Univ. School of Aviation Med., U.S.A.F., Randolph A.F.B., Tex. 56-118 (July) 1956.

XLIV

THE MEATOANTROTOMY — A SPECIAL APPROACH FOR PLASTIC PROCEDURES

Fritz Zöllner, M.D.

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First, an explanation of the term "Meatoantrotomy":

One differentiates the simple antrotomy or mastoidectomy in which only the mastoid is removed and the important functioning part of the middle ear is untouched. In the radical operation all of the diseased sound conducting mechanism is removed. If one performs a conservative radical operation or attic-antrotomy, the drum, malleus, incus and stapes are not touched but the attic in which the malleus and incus lie must be opened. If this area is not seriously diseased and needs only to be examined, or there is need for better accessibility to the tympanic cavity because the meatus is too narrow and does not permit a full inspection of the drum or its remains, then a special type of conservative procedure is necessary which I would like to name, as mentioned before, "Meatoantrotomy." By this method the mastoid cells are completely removed as far as the antrum and at the same time the bony meatus is opened in order to facilitate the examination of the drum. Because a wide bridge is maintained, the bony protection surrounding malleus and incus remains untouched. From the definition you see that indications for this operation exist, 1) in the case of a dry central perforation when the meatus is too narrow to close transmeatally or 2) the same defect with recurring flow of pus as a result of chronic changes in the antrum. The most successful and ideal form of plastic procedure remains the transmeatal closure of drum defects. This operation which was developed and performed in the last century by Tangemann has been rediscovered by Wullstein and independently used and modified by Juers, Frenckner and Pietrantoni. I have used it myself since 1953 many times and still use it if the ear is dry and the meatus is wide enough. In small defects the border of the perforation is cut away, the surrounding epithelium excised and a small Thiersch graft is

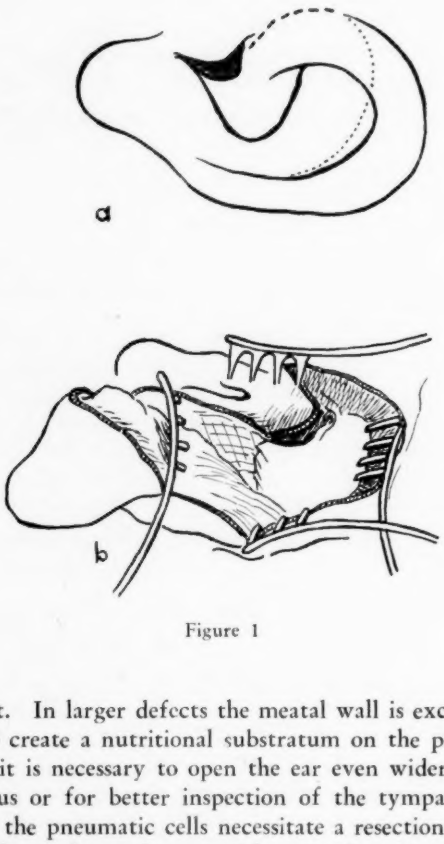

Figure 1

placed over it. In larger defects the meatal wall is excised, and it is also better to create a nutritional substratum on the promontorium. If, however, it is necessary to open the ear even wider because of a narrow meatus or for better inspection of the tympanic cavity, if conditions in the pneumatic cells necessitate a resection of the whole mastoid, we have always begun with an attic antrotomy. In the course of time we have become more conservative just as Wullstein, and have come to use the technique we call the Meatoantrotomy. The type of incision is basically unimportant; one can open the ear posteriorly or use the endaural way. For all operations in the tympanic cavity we prefer the incision, published by Heermann in Essen in 1939, around the upper part of the auricle (Fig. 1a). The first incision goes only as deep as the fascia of the temporal muscle. The ear is bent down and on the lower border of the muscle the incision is carried through the periosteum to the bone. At the same time

the meatal wall is incised to the level of the bone at 12 o'clock. Following this incision, a second vertical one is made in the posterior meatal wall. Now the ear can be bent down even farther. The planum mastoideum is open (Fig. 1b). When necessary, it is possible to expose the whole mastoid by pushing aside the soft parts. In this way the bony part of the meatus is easily accessible. We prefer the old method of incision, behind the ear, only when a large operative cavity in a case of extensive pneumatization must be made smaller by displacement of the soft parts. A short rubber tube which determines the size of the meatal opening is inserted before suturing. If the incision is done very precisely the cosmetic result is very good. It must follow exactly the internal border of the helix. If the ear is bent down in this way, the mastoid cells can be removed, and the antrum opened.

The aditus ad antrum should not be opened too wide in order not to expose the body of the incus. At the same time the outer two-thirds of the bony meatus is removed and a wide bony bridge which completely covers the auditory ossicles remains. In order to make the tympanic cavity more accessible, this wide bridge must be carefully drilled and thinned from the front. During this step it is practical to incise the meatal skin in order to be able to see into the tympanic cavity (Fig. 2). This can be done by two methods: first by building a tympanomeatal flap (b and d), by cutting at 12 and 6 o'clock. This flap on bending it back later serves to cover the aditus. This way of building a flap is especially efficient and elegant, but not easy for the surgeon. During the operation the flap covers the tympanic cavity and limits the view. The second method opens the meatus like the wings of a folding-door (a and c). It is easier and the view is better. If one drills away enough of the bone above and below the posterior meatus, both the flap-ends build a flat threshold in the cavity. I have fastened the top-flap with a suture. A small skin-projection remains on the posterior drum border (c). In order to examine the incudostapedial joint and to make certain that a cholesteatoma is not overlooked, the window-niches must be made accessible. The best way is similar to the way we do Rosen's mobilization. That is, to luxate the posterior drum border carefully (a), and, when necessary, enlarge the bony frame (b). One can also leave the frame intact and drill a control-hole in the wide bridge, as Wullstein has suggested. Through this small hole one can see both

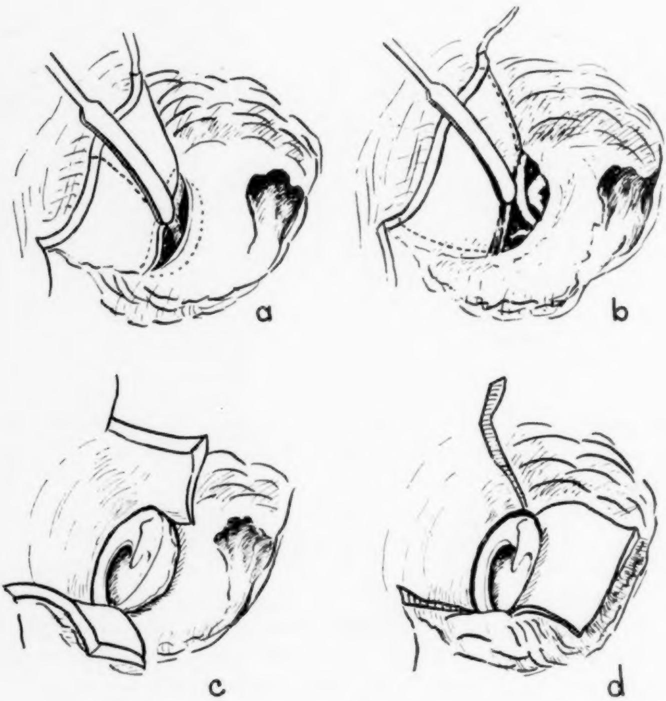

Figure 2

windows very well but, of course, an operation is not possible under these conditions.

If the examination reveals a movable and intact incudostapedial joint after opening the posterior drum border or looking through Wullstein's window, then one can perform a plastic closure. This is performed as in the transmeatal method by excising the perforation-border and removing the bordering epithelium from the remains of the drum or the meatus. In as much as we are dealing with larger defects it is necessary to select skin transplants somewhat thicker than Thiersch grafts, approximately 0.5 to 0.6 mm thick. One is

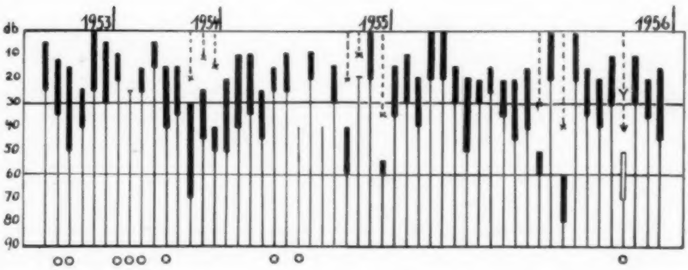

Fig. 3.—Number of operations, 52; Improved, 47 (90%); Reaching 30 db, 49 (94%).

assured of good results if the borders are carefully placed on the remains of the drum or the floor of the meatus. Since thick transplants are not easily adjusted to the curvatures, I use grafts with a thick center and thin border. Such grafts can be obtained with a little practice by lifting up skin-folds and cutting with the knife. By the use of an electrical dermatome it is also possible to obtain at least a small thin border which facilitates the fitting to the meatal floor. We do not insert an inlay in the tympanic cavity. The aditus ad antrum is either closed with a tympanomeatal graft or with a second transplant. All of the remaining cavity is covered with the thinnest possible skin-grafts.

The results of these operations are excellent. The hearing results are the same, whether one closes a drum defect transmeatally or a meatoantrotomy is carried out. However, the latter allows a wider selections of patients. In Figure 3 the hearing results are presented in 52 cases which I have operated upon since 1953. The thin lines represent the hearing before the operation, the thick lines the hearing-improvement, the empty spaces the hearing-loss, the dotted lines the hearing-loss in bony conduction before the operation. In 93 per cent of the cases there was a hearing gain of at least ten decibels.

Theoretically one should expect a normal or almost normal hearing after these operations. This was achieved only in 9 out 52 patients. This was caused by the adhesions and stiffening of the

auditory ossicles. A loss of hearing occurred in only one case in which a degenerated inner ear became worse. The small rings underneath (Fig. 3) denote recurrence of secretions. This occurred in the first group of cases more often than later as a result of false selection of patients. In many of these cases at least part of the hearing-gain was maintained. This can be explained by the fact that only a small part of the flap perforated again, so that in spite of a temporary secretion the function remained improved.

In general we may be very pleased with these results. We would be happy if there were more suitable cases which could be cured by a simple closure-plastic or meatoantrotomy.

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Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Thursday, November 8, 1956

(JOINT MEETING WITH
CHICAGO ROENTGEN SOCIETY)

THE PRESIDENT, DR. RAYMOND W. KERWIN, IN THE CHAIR

Malignancies of the Nose and Nasal Accessory Sinuses.

Clinical Diagnostic Aspects

ARTHUR LOEWY, M.D.

(Abstract)

The term "malignancies" in relation to the nose and nasal accessory sinuses embraces many possibilities. Malignant tumors may arise from any of the diverse components of the area—from mucosa, bone, cartilage, blood vessels, lymphatic elements, neural tissues, pigment cells, from embryonal rests and from other sources. However, review of the published data is in general agreement that, for practical purposes, malignancy in this area implies carcinoma, since 99 per cent of the histologically malignant tumors are carcinoma, and the great variety of other possibilities accounts for one per cent. Of course, as with all medical statistics, the patient with the "one per cent tumor" is 100 per cent involved by his infrequent tumor.

There are tumors of the nose and sinuses that are histologically benign, yet they exhibit clinical features generally ascribed to malignant tumors. Such a classification includes inverting papillomas, ossifying fibromas, chondromas, giant-cell tumors, and salivary gland type tumors. While these tumors do not exhibit the pleomorphism, anaplasia, or mitosis associated with the microscopic diagnosis of

malignant tumors, they do exhibit a marked tendency to recur when removal is incomplete. They erode and infiltrate adjacent tissue; they cause deformity and destruction, and may prove fatal.

Early diagnosis of malignant disease is always an important feature in improvement of salvage rates by any modality of therapy. It is certainly important in malignant disease of the nose and sinuses in view of the proximity of vital areas to which the tumor may advance in its relatively early stages. Clinical diagnosis is the fundamental means of case finding early or late, and depends upon evaluation of symptoms, examination by available means, and upon the histopathologic examination of properly selected biopsy tissue.

An evaluation of the symptoms most frequently reported as the presenting complaints indicates that these complaints are not unusual or unique. Some are nasal symptoms, some are general head symptoms, others may be referred to the eye or to the mouth, but none herald the dire consequences of an advancing, potentially fatal disease. It is significant that none of the symptoms can be considered as an early symptom, because each depends upon the erosion and invasion by, or enlargement of the tumor. Further, each of the symptoms is similar to the symptoms of a variety of benign nose, sinus, eye, or dental diseases. These symptoms are obstruction, discharge, bleeding, deformity, swelling and pain.

Intranasal examination does have its limitations. In spite of mucosal shrinkage or anesthesia, there are "blind" areas to both anterior and posterior rhinoscopic view. The electric nasopharyngoscope may serve to give a limited intranasal view behind septal obstructions or in other areas difficult to expose; the smaller calibre electric nasopharyngoscopes can also be introduced into the antrum by means of trocar puncture of the inferior meatus. Used in this way, the instrument cannot obtain a biopsy, but it may give support to indication for surgical exploration of the antrum by means of a sublabial antrotomy, in such conditions as intrasinus bleeding, bulging of the lateral nasal wall, or cheek swelling without break-through. Fundamentally, clinical diagnosis depends upon an awareness of the possibility of the presence of malignant disease, and the biopsy of suitable tissue.

The gross appearance of this group of tumors is an unreliable diagnostic aid. Intranasal tumors may invoke a polypoid response in the adjacent normal tissue or may themselves have polypoid fea-

tures. Erich has reported papilloma, fibroma, plasma-cytoma, hemangioma, pyogenic granuloma, and carcinoma all simulating polyps. Miller has related a series in which three per cent of presumed benign polyps demonstrated malignant change not clinically suspected. This type of evidence should demand histologic examination of all tissue removed.

Case presentations illustrate: a) the polypoid character of some tumors, b) the symptoms referred to other organs or areas—eye and dental, c) the resemblance between tumor and acute sinus disease, and d) delayed diagnosis based on delay in obtaining suitable biopsy material.

Evaluation of delayed diagnosis demonstrates that the delay may be based on the sometimes misleading symptoms as illustrated: symptoms that direct attention to other organs, or symptoms that resemble benign, self-limited disease. It may also be based on misjudgment or artefact at the laboratory level, where areas of edema rather than true tumor are sectioned, or where judgment is passed on the basis of examination of relatively benign areas within the bulk of the tumor. Thus, where clinical diagnosis of malignancy is suspected, a negative histopathologic report should not be regarded as a final pronouncement; repeat biopsies may be in order.

In conclusion, the fundamental move of clinical diagnosis in malignant disease of the nose and sinuses rests upon obtaining a satisfactory biopsy of representative tissue. This, in turn, depends upon an awareness of the possibility of malignant disease causing such common symptoms as obstruction, discharge, bleeding, deformity, swelling and pain.

Roentgenological Diagnostic Aspects

John H. Gilmore, M.D.

(Abstract)

The x-ray examination of the paranasal sinuses and nasal passages plays a very important part in the management of the patient with suspected neoplasm in these structures. From the x-ray findings one can very often tell whether there is a tumor present or not, whether it is benign or malignant, where it had its origin, and how far it extends.

Importance of tomographic study of the nasal passages was stressed with demonstration of a benign lesion, a rhinolith.

Numerous malignant neoplasms of the maxillary sinuses were demonstrated from very early findings to far-advanced. The technique of the x-ray examination was stressed. A balanced technique to show both soft tissue and bone detail is very important.

The appearance of abnormal density in the sinuses was shown. The density of a malignancy is greater than either polypoid degeneration of the mucous membrane, which has a ground glass appearance, or the variable density of thickened membrane and exudate. The only abnormal density of the sinuses greater than that due to malignant neoplasm is the osteoma.

The value of tomography in determining the extent of involvement of a malignant neoplasm beyond the sinus of origin was shown and how the point of "break through" can be identified.

In differential diagnosis conditions included were mucocele of a frontal sinus, numerous osteomata of ethmoidal and frontal sinuses with mention made of osteitis.

The demonstration was concluded by stressing the importance of examination of additional areas such as the chest which, at times, will give very important and deciding information as to the true nature of the existing situation in the sinuses in question.

Indications for Surgery

Irwin D. Horwitz, M.D.

(Abstract)

The treatment of a patient should not be conditioned by the often antagonistic proponents of different modalities of therapy.

Surgery alone is not efficacious in the management of cancer of this area. This has been our experience despite the excellent results reported by Ohhgren (38 per cent) and New (40 per cent). Ohhgren reported a series of 187 cases, 149 of which were treated electrosurgically. However, of the 20 patients with a five-year survival,

15 only had a carcinoma, while four had a sarcoma, and one a mixed tumor. This gives a cure rate of 10 per cent for carcinomas. The objection to New's statistics, and not to the validity of his work, is that he selected his cases, rejecting almost half of the patients as unfit or unsuited for surgery. In the cases to be reported, all patients registered are considered, even those so morbid that they died before therapy could be finished.

Hendrick in a recent article states that neither surgery nor irradiation alone is satisfactory. He followed a method of therapy similar to ours, but differing in that the irradiation was given before surgery. His series is too recent to include five-year salvage rates, but his three-year salvage rate is excellent—14 of 19 patients living.

The plan of treatment followed by us at the University of Illinois is adequate surgery followed by cancerocidal doses of irradiaation. Any patient with a unilateral cancer that has not invaded the base of the skull and has not metastasized below the clavicles is considered operable. If there is any suspicion of orbital involvement, the contents of the orbit are exenterated and the infra-orbital plate removed. One-half of the hard palate is always removed. The ethmoidal labyrinth is exenterated (and usually electrocoagulated), the maxillary sinus walls are all removed, extending as far posteriorly into the pterygoid fossa as necessary. We have not appended any man's name to the procedure (Hautant, Moure, Fergusson, etc.); any other surgery is considered inadequate and the Caldwell-Luc and Denker operations are mentioned only to be condemned as inadequate for tumor surgery.

Immediately postoperatively irradiation is begun, either intracavitary if the lesion seems limited, or external if the tumor seems widespread.

Since 1954, 99 cases have been registered in the tumor clinic. Of these all but five have been carcinomas, and of these five none have survived. Two were lymphosarcomas, one a melanoma, one a salivary gland tumor, and the last a fibrosarcoma.

Of the cases, 57 were treated by surgery and irradiation, 41 by irradiation alone, and one, far advanced and hopeless, by an experimental serum. There are to date 18 five-year survivals, 16 have been treated by the combined method, two by irradiation alone. However, there are six cases that are free of disease for a period over three years,

but less than five; these have all been treated by the combined approach. This gives a five-year salvage rate of 18 per cent and a projected five-year survival, counting those cases who have been free of disease over three years, of about 24 per cent.

What does not show in the statistics, and what we think is vitally important, is that surgery gives immediate palliation from pain and fetor. The patients stand the surgery remarkably well with little morbidity and no mortality. Age is the least barrier; of the 57 cases, 22 were over 60 years old. Rehabilitation in most cases is relatively simple with a suitable prosthesis closing the palatal defect.

To summarize, patients with a malignancy of the paranasal sinuses have a better chance for a cure if treated by a combination of adequate surgery and cancerocidal doses of irradiation.

Indications for Surgery

W. M. Ironside, M.D. (by invitation)

(Abstract)

It is as well to reaffirm the principle that all soft tissue be dealt with by electrosurgery except when incising skin. Surgery in this field serves four purposes:

1. Diagnostic—to obtain a biopsy and to estimate or confirm extent of lesion.

2. Drainage—to allow escape of pus and degenerate tumor following radiotherapy.

3. To provide an observation window in cases involving the sinuses.

4. To eradicate the malignant lesion—either alone or combined with radiotherapy.

Diagnostic. Although cytological studies may be of help in the diagnosis of these lesions, everyone is agreed that confirmation by biopsy is essential, and to obtain a good biopsy in suspected sinus lesions, adequate exposure is necessary.

Drainage. There is little argument that some sort of drainage procedure is necessary before the start of a course of radiotherapy for

a malignant lesion of the sinuses. This allows the escape of purulent material so often associated with tumors in this site and degenerate tumor tissue following radiotherapy.

As regards providing an observation window, it is becoming more and more apparent that this is necessary in sinus lesions in order to provide an adequate follow-up after treatment. To await a recurrence within a sinus to declare itself by presenting at an antrostomy opening or as an extension into the cheek or eye is not acceptable today. The two sites usually used for these openings are the sublabial region and palate. The site chosen depends on the original site of the lesion and the treatment given. If the lesion has been largely palatal and surgery used to deal with it, then a palatal window is the choice. If, however, the sublabial approach has been used, then here is the obvious site for the window. These windows should be made at three to four weeks after the completion of a course of radiotherapy. It is possible at this time to check the result of the radiotherapy and deal with remaining malignant tissue.

As to the fourth point where surgery is used to eradicate the lesion, the problem is more complex.

In the brief time allotted it would be convenient to tabulate that indications for curative surgery, but with malignancies of the nose and sinuses it is impossible and would be misleading. All that can be hoped for is a few guiding principles.

In malignancies of, for instance, the larynx which, fortuitously, is also on the program, the problem is not so great. In this site the type of lesion is nearly always the same histologically, the site of the lesion is nearly always readily and early visible, and the lymph drainage field accessible surgically. Bearing these points in mind, and having considered one's own experience and the results obtained by various methods at other centers, it is possible to be more definite about indications for curative surgery.

Considering the points just mentioned as applied to the nose and sinuses:

A. *Type of Growth.* More than two-thirds of the malignant growths in the nose and sinuses are carcinomatous. The majority of the remainder are sarcomatous. Some of these sarcomata are very radiosensitive, and a five-year cure rate of 50 per cent has been claimed

in a small series, with radiotherapy alone. Certainly surgery can offer no better results, and it has been suggested that it may even be dangerous. So, in these cases, surgery is only of benefit to obtain a biopsy and provide drainage and an observation window. With carcinomata and the more radioresistant tumors, most authorities agree that the best results are obtained with a combination of surgery and radiotherapy. The results which can be obtained by combined treatment are about 30 to 35 per cent five-year cure (Wilson and Windeyer and Capps). This is an advance on either surgery or radiotherapy alone. As an example of the results obtainable by radiotherapy alone, let me present a short series treated in Edinburgh.

They received one of three forms of treatment: 1) intracavitary radium; 2) deep x-ray; and 3) a combination of 1 and 2. Of the 35 patients in this series, eight, or 23 per cent, were alive after five years. It may be of interest to the therapists present that the only case to be treated through wedge filters gained a five-year cure. The problem, however, is to decide which form of treatment should be offered first.

On reviewing the literature, there appears to be little difference which is given first, the results obtained being the same. As a result of the extensive necrosis and delayed or incomplete healing which occurs following radiotherapy of lesions which have involved the skin, in these cases it is probably a sound practice to operate first and follow up with radiotherapy. As a result of brief experience of a very limited series treated with a 4 mv accelerator, it is felt that more cases should have this form of therapy primarily. With these machines the surface dose is low and so the problem of skin necrosis does not present so frequently. Again, as a result of low surface dose, it is possible to irradiate through the open eye without inducing eye complications seen in the past with older methods. Furthermore, the beam is more deeply penetrating and so the tumor area can be more accurately irradiated. As already mentioned, this is a very short series and it is far too early to give any definite figures, but early results are promising.

It might be convenient at this stage to consider the advisability of excision of the maxilla as a planned procedure. So often it is unnecessarily radical and frequently not radical enough. It is felt that when embarking on surgery to eradicate the disease, there should be no set procedure in mind. Once the route has been decided, depending on the site and extent of the lesion, one should aim at removing the tumor

with a margin of healthy tissue as far as possible. While excision of the maxilla may occur while doing this, it is considered a mistake to start off with this in mind.

B. *The Site.* The site of the growth also influences the decision as to what treatment to offer. In localized lesions of the septum it is probably wise to offer surgery as there is a fair danger of cartilage necrosis with radiotherapy.

C. *Lymph Drainage Area and Its Accessibility.* Unfortunately, most of these tumors tend to drain into the retropharyngeal lymph nodes. As a result, it is exceedingly difficult, if not impossible, to remove these involved nodes by surgery and radical neck dissection is not likely to meet with much success.

On the other hand, it is as well to remember that the results obtained by radiotherapy in dealing with cervical metastasis are not very cheering.

Surgery, of course, plays a large part in the treatment of recurrences at the primary site, as a second course of radiotherapy is generally impossible if the first course has been adequate, and local radium has only a limited use in these cases.

In conclusion, in all cases surgery is necessary for taking a biopsy and in all sinus lesions for providing drainage, and an observation window. When to eradicate the lesion by surgery is a more complex matter, and it can be appreciated that the decision can only be made by a closely cooperating team consisting of at least the surgeon, radiotherapist, and specialist in prosthetic work.

Indications for Irradiation

Marion F. Magalotti, M.D.

(Abstract)

The treatment of cancer is difficult anywhere in the body. Cancer of the paranasal sinuses is especially difficult because of their anatomy, that is the predominance of bone and the scarcity of soft tissue, the latter being chiefly a thin lining of mucous membrane from which most of these cancers arise. The bone acts as a barrier to the

extension of the cancer but does become infiltrated with tumor, long before the latter is demonstrable on an x-ray film. Primary carcinoma of the frontal and sphenoid sinuses is uncommon; the antrum is most commonly involved and the ethmoids second.

The occasional adenocarcinoma and primary bone tumors are best handled by surgery. Lymphosarcoma and lymphoepithelioma are very radio-sensitive and best handled by radiotherapy. The overwhelming number of malignant lesions in this region are squamous cell carcinoma. The management of these cases should be decided with the mutual cooperation of the otolaryngologist and radiotherapist. Under no circumstances should radiation therapy alone be chosen with the idea that it is easier for the patient. That is incorrect. There is a definite morbidity associated with adequate therapy in this region aimed at a cure. The discomfort of radiation reaction, tumor and bone slough, secondary infection, and finally sequestration cannot be denied. Of course, the surgeon has his problems, too.

Larsson and Martensson consider the following to contra-indicate surgery:

1. Advanced age.

2. Poor general condition or complicating diseases having a doubtful prognosis.

3. Roentgenologically demonstrable destruction of the base of the skull or of the pterygoid process.

4. Very extensive bone destruction or considerable bilateral spread.

5. Extensive involvement of the soft tissues of the cheek or the external nose, where radical operation would entail large and disfiguring defects.

6. Tumorous infiltration of the mucous membrane in the nasopharynx.

7. Inoperable lymph node metastasis.

8. Generalized metastasis.

9. Refusal by the patient and in severe mental disturbances that would make a defective condition difficult to overcome.

In our department, the management of a case is decided in cooperation with the attending otolaryngologist. The decision may be

surgery, external radiation or intracavitary therapy, or a combination of these. Patients with cancer may be in a serious state of malnutrition, with secondary infection and anemia. Efforts should be made to improve or correct these deficiencies, before beginning specific therapy, whether it be radiation or surgery. The administration of antibiotics, parenteral and tube feeding and blood transfusions are of vast importance in preparation for surgery, but how often it is forgotten in the preparation for radiotherapy.

A few cases will be briefly reviewed to illustrate the various methods of irradiation which we use.

REPORT OF CASES

CASE 1. A 21 year old Negro female was seen in November, 1954, with the history of progressive swelling of the left cheek, periodic nose bleeding and difficulty in breathing. The clinical examination revealed a mass within the left nasal fossa arising from the lateral wall. The x-ray film showed cloudiness of the left antrum with osseous changes of the medial wall. The biopsy report was squamous cell carcinoma. A plaster cast is seen of the patient's face which was used to prepare the lead mask. Also seen is the lead mask which was swaged from a 2 mm sheet of lead. The two 5 cm in diameter anterior openings and one 7 x 7 cm lateral opening can be seen in the mask which served as ports of therapy. The appropriate angles were determined to direct the beam from each port. A tumor dose of 5000 r was delivered in six weeks to a volume of tissue which encompassed the tumor, with 200 KV H.V.L. of 2 mm Cu and 50 cm distance. There was a gradual slough of the left inferior turbinate and a portion of the medial wall of the left antrum. The patient did well clinically and was instructed in the daily cleansing of the left nasal fossa. In March, 1956, a sequestra was removed under general anesthesia from the left nasal fossa.

This attractive young girl left Chicago several weeks ago before I could get a photograph for this meeting. She joined her husband who is in the service in Japan. She has been fine with no prosthesis and no evidence of disease approximately two years after external therapy.

CASE 2. A 19 year old colored female that was first seen by the ear, nose and throat clinic in October, 1950, complaining of bleeding from the right nostril. A large polyp was found arising from the

floor of the right nasal fossa. This was excised and the pathological report was basal cell carcinoma. The patient did not return for ten months. At this time, the entire right nasal fossa was filled with tumor. As shown on the slide, x-ray therapy was given through two anterior ports delivering 3600 r at a depth of 4 cm. Following x-ray therapy, the right nasal fossa was emptied surgically. Residual disease was found on the floor of the nasal fossa extending up to the lateral and medial wall. The rebiopsy now showed squamous cell carcinoma. As shown on the slide, radium therapy was given with a single tandem, at two sittings, delivering each time 2500 gamma roentgens at the periphery of a cylinder, 2 cm in diameter, within one week's time.

A gradual slough of the floor of the nasal fossa with the nasal septum and lateral wall followed, requiring a prosthesis for the hard palate. The patient left the city and was seen again two and one-half years later. In August, 1955, a large sequestrum was removed under general anesthesia which involved the central portion of the maxilla, the floor of the nasal fossa, and portion of the nasal septum. No tumor was found. This photograph taken recently shows the patient, now age 24, five years after combined x-ray and radium therapy with no evidence of tumor. She wears a prosthesis.

Case 3. This case illustrates a combined management of surgery, x-ray and radium, in that order. A 52 year old white male was seen in June, 1955, with a history of surgical exploration and partial antrectomy two months previously. There was obvious tumor on the walls of the amphitheatre and the pathological report was anaplastic squamous cell carcinoma. X-ray therapy was started and 2100 r (air) was given through a 7 x 7 cm anterior port, and the same amount through a similar lateral port in three weeks with 200 KV H.V.L. 2 mm Cu and 50 cm distance. This was followed immediately with intracavitary radiation.

Several types of intracavitary sources are shown for the nasal fossa, antrum and surgical amphitheatre. The smallest source is used for the antrum, being inserted directly into the antrum through a small opening in the supra-canine fossa, as advised by Dr. Paterson of England. The source on the right was used in the nasal fossa of the second case we discussed. The two plastic-covered sources are used for intracavitary therapy within the surgical amphitheatre, the size used depending upon the volume to be treated. The smaller plastic-covered source was used in this third case. It was introduced with no anesthesia, being held in place with a sponge.

The film shows the plastic-covered source in the surgical amphitheatre, the visible portion being the brass tube holding the cobalt sources. This source was left in place for six days, delivering 6000 gamma roentgens to the periphery of a 5 cm diameter sphere.

This patient recently returned after a six months' absence. The skin has retracted and exposed a portion of the maxilla. Surgical resection of this is being planned. The surgical amphitheatre is clean with no evidence of tumor, 18 months after radiotherapy. None of these cases developed cervical metastasis.

In conclusion, we know there is much to learn about the management of cancer. The ideal treatment will be found some day in the future to replace our rather primitive methods, but until that day arrives, let us approach these unfortunate patients as a team, with prejudice in only one direction, against cancer!

Summary

SAMUEL J. PEARLMAN, M.D.

(Abstract)

From the presentations you have had, it is fair to say that the criteria for the treatment of malignancies of the nasal cavities and their accessory sinuses are not rigid. Treatment may be limited to surgery alone, to radiation therapy, before and after surgery, and to radiation therapy alone.

The latter may be in the form of x-rays, or radium at a distance in the form of the bomb, or radium locally.

Schuknecht says some neoplasms in this area are so radiosensitive that they may be treated by radiation therapy alone. He says that surgical resection should not be attempted when there is no reasonable chance for complete removal of the neoplasm. "The criteria for operability are determined to some extent by the surgeon's skill and courage."

He indicates the contraindications to complete surgical removal and names among them invasion of the skull, nasopharynx bilateral involvement and distant metastasis. He thinks that 30 per cent

about of all patients with carcinoma of the accessory sinuses are inoperable and should receive palliative radiation therapy.

Lymphatic involvement offers some food for thought. It is apt to be late; resection of the nodes when involved must of necessity be discontinuous, thus violating the principle of removing the tumor and its lymphatic supply en masse.

In this connection, it is of interest to know that Lyall considers the neck one of the few areas in the body where efficient node dissection can be carried out.

The commonest early symptoms of benign and malignant growths in the area under discussion you have been told are obstruction to breathing, nasal discharge, and nosebleed. These symptoms occur in other diseases too. Other diseases are many, and carcinomata are relatively few; still these obscure symptoms in persons usually past 50 call for the greatest scrutiny until they have been clarified.

In line with this thought, it has long been known that cancers differ markedly from each other, as witness just the variety met with in the series of 72 cancers of the nasal fossae and paranasal sinuses reported recently by Hendrick; he names squamous cell carcinoma, adenocarcinoma, malignant melanoma, lymphosarcoma, lymphoepithelioma, myxosarcoma and cylindroma. The manner in which these various types develop, their rate of growth, their invasiveness, and the way in which they spread to lymphatics and to distant parts of the body must and does vary greatly.

Even the concept of what is early and what is late is hard to define. Certainly there are patients who delay their coming; there are physicians who delay their diagnosis, and there are a large number where diagnosis simply cannot be made "early" in any sense of the word.

Lastly, in carcinoma of the maxillary sinuses the site of origin within the confines of the cavity determines the patient's fate; in one area where the orbit or the skull are invaded early, the prognosis is worsened, in another, the site remains more confined and yields more easily to treatment.

The tendency to increase the scope of operation for carcinoma is not limited to this area, it applies here as it does in other areas of

the body and in recent times has come in for some critical evaluation. It is fair to point out that the tendency to radicality is not due altogether to an increase in surgical skill, but rests in large part on all those factors which have made all surgery more successful in recent years. These factors are, as you all know, dependent upon better anesthesia, blood loss replacement, control of infection by antibiotics, restoration of body fluid loss and electrolyte balance as well as improved building-up of the patient's body by proper diet, vitamins, etc.

These surgical procedures cannot be done by the larger number of laryngologists, well trained though many of them are nowadays. The treatment for these conditions must, in large part, remain in the hands of those few who have the opportunity to become most expert and whose duty it is as teachers to bring the rest of the profession up-to-date over the necessary period of time.

Here I quote Zinninger: "Regarding the question of morbidity, deformity, or disability of the patient following supra-radical operations, there is considerable difference of opinion among both surgeons and patients. Most of them would like to eat their cake and have it too. The patient, particularly, does not want a disability or deformity but he does wish to be cured. The surgeon here needs to take the initiative. The patient is a layman who will in all probability be guided by the surgeon's advice, and the latter can 'sell him' or discourage him from a given course. The surgeon should not take advantage of his knowledge to convince the patient that a big operation is better for him than a less extensive one unless there is clear-cut evidence of the superiority of one operation over another. Patients who become partial outcasts because of disfigurement, invalids because of inability to eat or properly digest food, or who suffer long periods of disability before they finally can resume more or less normal activities, may ultimately feel that they have paid a high price for the privilege of living unhappily."

To the above may be added the odor of bone injured by radiation therapy or electrosurgery and which may not disappear for the longest time and only after complete sequestration has taken place.

"Different procedures each present different hazards and challenges. Not infrequently, of course, the operation may change an apparently hopeless cripple into a self-respecting and useful member of society, but surgeons must remember that the opposite effect may also occur, and must, therefore, exercise their most mature and careful

judgment based on well-documented evidence. On the whole, while it is recognized that morbidity and crippling of one sort or another may follow these extensive operations, in many instances it is not serious a problem."

In concluding, it must be emphasized again that there is no sole and proper treatment, each case must be individualized; where possible, a cure by lesser means is preferable, and the physician must maintain a constant conscientious revision of method.

There is certainly no intent on my part to preach a form of therapeutic nihilism, nor do I mean to decry the increasing scope of the modern approach. In this connection we must agree with Lyall who says, "An attempt to improve a procedure is not an admission of failure, but rather it is the duty of all physicians."

Betatron Therapy in Carcinoma of the Larynx

Lewis L. Haas, M.D.

(Abstract)

My role is joining the symposium to discuss in general the possibilities of betatron therapy in cancers of the nose and throat area, its relation to conventional x-rays, and to show a few irradiated cases. The main question is what is the sense of using megavoltage radiations in lesions which are close to the surface and to which conventional x-ray may also deliver an adequate, high tumor dose.

The betatron may produce two types of radiation: high energy x-ray beam and high energy electron beam. Their depth dose distributions, and accordingly their indications, are entirely different. In the curve of conventional 400 KVp the maximal dose is at the surface, the decrease is fast, so that at 10 cm depth the dose is less than 40 per cent of the maximum. In the curve for the 23 MeV betatron x-ray beam, the dose is insignificant at the surface, it remains relatively low in the first 3 cms, reaching the maximum at 4 cm depth. The decrease is slow so that in 10 cm depth it is still 80 per cent of the maximum. The curve for the high energy electron beam shows that the absorption is rather homogeneous up to a certain depth, and beyond it the dose drops down rapidly.

The possible advantages of betatron x-rays above conventional x-rays are: 1) greater penetration, a higher tumor dose can be delivered to any depth of the body, 2) lower surface dose, therefore a skin damage cannot be expected even after excessive dosages, 3) insignificant side scattering, sharper beam limitation, therefore normal tissues outside of the beam receive no radiation, and 4) less bone absorption, therefore the risk of bone necrosis and bone marrow depression is less.

The indications for betatron x-rays may be divided into three groups: 1) the main indications are deep seated lesions, because a higher tumor dose can be delivered with less general, less skin and bone reactions and damages, 2) potential indications are less deeply seated lesions to which conventional x-ray also may deliver an adequate tumor dose, but the risk of skin and bone damage is less, and the volume of irradiated normal tissue is less, 3) spray radiation of large body parts or of the whole body for palliation in extensive or metastatic conditions. The betatron secures more homogeneous absorption in large body parts, higher dose in all lesions, and less bone marrow depression. In the nose and throat cancers, only the second group of potential indications is of interest.

In treatment of sinus carcinoma the betatron x-ray beam would have advantages and disadvantages. The advantages would be: less risk of skin reaction and bone damage, and in lack of side scattering, diminished risk of eye damage. Disadvantages are: the high exit dose, and mainly the low surface dose, because tumor segments which are close to the surface in advanced cases would receive an insufficient dose. In our material we had no opportunity to treat sinus cases in which the betatron x-rays were preferable to conventional x-rays.

More ideal is the dose distribution of the high energy electron beam. Its advantages are: 1) rather homogeneous absorption to a certain depth, therefore the tumor regression is more uniform and gradual in all layers of the tumor, 2) beyond this depth the dose drops down rapidly, therefore the nearly normal tissues will get much less radiation, and the reactions will subside faster and better than after conventional x-rays. The depth of useful penetration can be selected as desired in the individual cases between 3 and 8 cm from the surface at our energies, 3) less bone absorption, therefore the risk of bone and cartilage damage is less.

The demonstrated slide shows, e.g., that the mucositis of the tongue stops in the midline in the selected depth. These advantages

are valid for the great majority of sinus carcinoma cases, without disadvantages. If our electron beam is available, we prefer it in sinus cases above conventional x-rays. The skin reactions to the electron beam are similar to those from the conventional x-rays.

In the first case a widespread postoperative recurrence of an adenocarcinoma of the right antrum was present, which was in part surgically excised. The extensive residual tumor included also the oral surface of the surgical palate defect. After electron beam treatment the tumor disappeared entirely.

The second case was a neglected epidermoid carcinoma involving both maxillary antra, nasal cavity, palate and vestibulum, with antro-rhino-oro-vestibular communications. He was treated for ten months with the diagnosis of palate ulcer. Because the depth of the lesion exceeded our maximal 8 cm penetration, we treated it through two opposing fields.

I wish to touch here upon a controversial problem, being together with nasolaryngologists. It is a traditional belief that the results of radiotherapy are better if inoperable large antral sinus carcinomas are reduced in size by partial resection. This was true perhaps in earlier eras when radium or lower voltage x-rays were applied with inadequate penetration. Since the use of higher energies, 200 KV or more, with adequate depth dose, this practice is obsolete. The impression is rather that the preradiative tumor amputation—except for special indications—impairs the chances of the patients due to several factors. In the delay of several weeks or months due to surgery, the disease may progress significantly or it may invade the vascular and lymphatic pathways of generalization. The incomplete surgical procedure may inoculate cancer cells into the vicinity and into the metastasizing pathways. We have seen repeatedly that after incomplete removal the tumor burst forth suddenly and the downhill course became more rapid. Surgical derangement of tumor tissue and tumor bed, and postoperative infections may impair and complicate the effects of radiations.

The localization of the larynx is a fortunate one, inasmuch as it is easily accessible to adequate dosage by any kind of irradiation, including conventional x-rays, betatron x-rays and betatron electron beam. The betatron x-ray has the advantage above conventional x-rays that the skin reaction and risk of cartilage necrosis is less. The electron beam has the advantage that the risk of cartilage necrosis is

less and that the mucosal reactions recover faster and better than by conventional x-rays. Therefore, we prefer, if available, the betatron x-rays above the conventional x-rays, and the electron beam above the high energy x-ray beam.

We have treated ten larynx cancer cases, seven of them refusing mutilating surgery, six were treated by x-ray beam, four by electron beam of the betatron. Three of the ten patients died, all of the third stage. Two were treated in the first period of our betatron work. One died of recurrent disease with neck nodes; he refused any further medical procedure, except for an emergency tracheotomy. We know now that the given dose of 6,300 betatron r in 45 days was insufficient. The second patient expired with symptoms of postradiation edema, also after tracheotomy, residual tumor was not proven, but it could not be excluded. In this case the given dose of 9,025 r was delivered for technical reasons in 32 days, perhaps too fast. The third patient died at the age of 85 years seemingly without local recurrence after three and one-half years. The out-of-state death certificate stated lung carcinoma and pneumonia as cause of death.

Seven patients are living without tumor evidence, one for over five, one over three, three over two years, one over one year, and one over six months, respectively. Using the classification of Nielsen-Strandberg, three patients belonged in the II stage, six in the III stage, and one with neck nodes in the IV stage. In the latter case radical neck dissection was performed in the meantime.

In the majority of cases without neck nodes small radiation fields of 3 cm diameter were used, and proved to be satisfactory. It is known that neck nodes develop later only in a small, less than 5 percent, percentage of the cases which can be cured by surgical dissection or radon seeding. The small fields give the possibility of larger tumor doses with less radiation reactions and damages.

In conclusion, in spite of the fact that conventional x-ray may also deliver an adequate curative dose to carcinomas of the sinuses and larynx, the x-ray beam and electron beam of the betatron megavoltage irradiation have definite advantages above conventional x-rays not so much in their curative aspect, as rather in lessened discomfort and less radiation damage. What the decreased discomfort during and after radiotherapy, and the lessened risk of skin, bone and cartilage damages mean clinically for the patients, the observing clinicians should express their objective opinion. Doctor Ratko who observed clinically the majority of our cases, will do it in his discussion.

Betatron Therapy of Laryngeal Carcinoma

ARTHUR L. RATKO, M.D.

(Abstract)

Since Dr. Haas has discussed the physical and radiotherapeutic aspects of this subject, my remarks will be concerned with clinical impressions obtained from observations of nine patients, so treated.

It would be pertinent at this time to briefly describe the classification we have used, namely, the Swedish classification.

1. Anterior two-thirds of one true cord.

2. Either bilateral cordal involvement or extension across the anterior commissure.

3. Extension beyond the true cords, fixation of a cord, no adenopathy.

4. All other lesions.

Of the nine cases the distribution is as follows:

1. None. 2. Two. 3. Six. 4. One.

Without going into the frequently debatable argument of radiotherapy vs. surgery, I might say that evaluation of all cases either by attending physician or the tumor board resulted in a recommendation of total laryngectomy in every case with one exception, which I will discuss later. In other words, it was felt that the anticipated cure-rate in these cases would be higher if total laryngectomy was done disregarding any question of function, preexisting disease, etc. Certainly, one could not anticipate an 80 to 95 per cent cure-rate which could be obtained with either modality of therapy in an early lesion as seen in Class I.

Our particular interest in these cases was centered in evaluation of three factors: A) course of the patient and evaluation of the tumor during therapy; B) posttreatment sequellae; and C) end result.

A. In the past, using conventional 200 KV therapy, the physical well-being of the patient has been compromised by several deleterious effects of radiotherapy.

1. Edema of both the airway and hypopharynx not infrequently precluded adequate respiratory exchange and/or nutrition. While tracheotomy, tube-feeding or gastrostomy were not usually necessary, using conventional therapy, their occasional necessity increased the morbidity and discomfort to the patient. Then, too, there was the occasional patient who required a permanent tracheotomy due to edema of the glottis which never subsided.

2. The hyperemia and edema seen during conventional therapy all too often made it extremely difficult if not impossible to adequately follow the lesion carefully, either during or after treatment. It is from these cases, I believe, that the highest incidence of failure of radiotherapy comes, since we cannot see that the lesion is not responding adequately.

B. With modern techniques of fractionation, multiple ports and adequate filtering, radio-necrosis of the larynx, or for that matter, the overlying skin, has become rare. This, indeed, is fortunate since I know of no more distressing situation than to obtain a good therapeutic result, sparing the larynx, only to have to resect it later because of the severe pain and debilitation which results with subsequent radionecrosis. One other sequela, which we have not been able to eliminate, is the endarteritis following therapy which increases the hazard of surgery considerably despite opinion to the contrary.

C. While the lesions in the cases under discussion were largely of the superficial variety, their location, particularly posterior extent, would yield a five year cure-rate of roughly 50 to 60 per cent if total laryngectomy were done. With radiotherapy we would anticipate a lesser cure-rate, particularly the case with adenopathy which we feel only rarely can be sterilized with radiotherapy.

Rather than give charts and statistics, which all too often are misleading, I would like to discuss these cases under the three headings above.

A. (1) In contrast to the hyperemia and edema seen within the first seven to ten days with conventional treatment, they appeared generally between the second and third week with betatron therapy. We chose to grade the reaction as slight, moderate or severe. In seven cases the edema was only slight, one case moderate, and one severe. In all cases but one, the edema subsided almost entirely within two to four weeks after therapy. Hyperemia, however, ranged from moderate to

intense in all cases particularly the hyperemia of the lesion itself, so much so that the lesion usually stood out quite clearly from the adjacent mucosa. In all but two cases the hyperemia subsided so as to leave only minimal telangiectasia.

(2) As previously stated, the intense hyperemia seen in most of the lesions made it simple to follow the course of the lesion. Because of the very limited edema, visualization of the lesion was excellent. It should thus be possible to predict, with reasonable accuracy, which lesions are responding favorably to therapy justifying its completion, or those in which radiotherapy should be discontinued and surgery carried out. In one of these cases (G.B.) the cord was entirely smooth approximately four weeks after therapy was begun.

No case required tracheotomy or supplementary feeding measures during therapy.

B. Since the skin receives the smallest dose in betatron therapy, there were no cases with significant skin damage either during or after therapy. In only one case (W.F.) did we feel that radio-necrosis had been produced. This was one of our earlier cases who received 10,000 R.T.D. in only 40 days, and he died of a combination of radio-necrosis plus persistent tumor three years and five months after therapy. One patient (I.L.) required tracheotomy four months after completion of therapy and died one month later with no information being obtainable with regard to either persistence or radio-necrosis.

C. These cases treated ranged in age from 32 to 81 with the majority being in the fifth and sixth decades. There was one female and eight males. Three patients have expired—one with known radio-necrosis and persistent tumor, one due to carcinoma of the lung, and the third cause of death is unknown. Five patients are alive and free of disease from 13 months to six years with betatron therapy only. The sixth case, of considerable interest, I would like to discuss briefly.

J.N., age 75, chief complaint, hoarseness and dysphagia for one month. Mirror laryngoscopy revealed ulcerative lesion posterior one-third at cord with extension to the arytenoid and partial joint fixation; firm, movable node, left neck. Diagnosis: epidermoid carcinoma. Treatment: 1. Surgery contraindicated because of I-V heart block and called extremely poor risk for monobloc resection of neck and larynx. 2. 8600 R.T.D. to larynx and neck node separately, April 7, 1954, and May 27, 1954.

Course: Laryngeal lesion disappeared entirely as did node. October 4: Wedge biopsy with radon seeding of recurrent nodule at neck, recurrent carcinoma. Left neck dissection on November 15, 1954, two tumor nodules showed central necrosis but persistent peripheral islands of tumor. Had breakdown with partial slough of upper skin flap which healed within three months. Patient free of disease and last seen September 5, 1956.

Here it was possible to eradicate a lesion by a combination of therapy with an apparently excellent result. It might also be said that the motility of the arytenoid is now normal.

While the small number of cases presented should not lead to generalizations, certain facts are readily apparent.

1. There is a marked reduction in the undesirable aspects of radiotherapy both during and after therapy.

2. There is a significant reduction in posttreatment sequelae.

3. The over-all results compare favorably with results of either surgery or conventional radiotherapy, perhaps are even better.

Abstracts of Current Articles

EAR

Testing "Cortical" Hearing in Temporal Lobe Tumors

Bocca, E., Colearo, C., Cassinaie, V., and *Megliavocca, F.*: Acta Otolaryngol. 45:4 (July-Aug.) 1955.

Eighteen patients with temporal lobe tumors in whom the pure and speech audiograms were normal were tested by live voice distorted by low pass filtration (1000 cps). These showed that the discrimination of the distorted voice is poorer in the ear contralateral to the lesion. In those in which the test gave negative findings exploration often revealed no involvement of the auditory cortical area and in these the test became positive after the surgical removal of this area. The authors suggest this test as of value "to explore the activity of the terminal stations of the acoustic pathway."

HILL

Hearing for Speech in Children: A Verbal Audiometric Test

Meyersen, L.: Acta Otolaryngol. Suppl. 128.

This monograph of 105 pages includes a discussion of the various methods of testing hearing in children and presents an original procedure for use with children. The author states that, while present techniques permit satisfactory study of the auditory capacity and function in adults, these fail to record similar precise evaluations in children.

In the infant group, three years or under, present methods yield conflicting results or only relatively gross data. While the use of the conditioned electrical potentials, as in the G.S.R. test, holds promise, much remains to be done before this technique is of practical application. A precise quantitative objective measure, not requiring voluntary response, is a requisite for making possible a positive diagnosis of deafness at this age.

In the older group, above three, he states that pure tone testing presents many difficulties due to the psychological factors involved, such as inattention, fatigue, or boredom. The validity of sweep check tests is open to question. The fading digit test frequently used for group testing in schools, even when used individually, is prone to errors. While speech hearing is preferable in children, merely substituting speech stimuli for pure tones is not sufficient.

Hearing for speech is a complex psychological function. Perception of speech and of pure tones in children are independent functions. Consequently speech hearing tests have a greater validity than pure tone testing and are of greater importance to the psychologist. The author agrees that pure tone testing is of value to the otologist, that acuity can be tested over the entire range of frequencies, that this can be correlated with acuity for speech, that it can be an indirect indicator of aural disease and that it yields diagnostic and prognostic data of importance. However, from the clinical and social psychological standpoint, a direct measure of hearing for speech is preferable. It was found that children with impaired hearing for pure tones usually had significantly impaired hearing for speech but conversely the majority of children with significantly impaired hearing for speech did not show similar impairment for pure tones.

The "verbal audiometric test," presented by the author, is the result of extensive systematic preliminary experimentation, carried out at the Psycho-Acoustic Laboratory at Harvard and supplemented by field studies in various schools in different parts of the country. Recorded voice is used. The reproducing equipment is very similar to that used by many otologists for speech audiometry (such as the Grayson-Stadler) except that in group testing (five or more) one earphone is a dummy to minimize ambient noise. Words of spondaic stress pattern were selected and equated for homogeneity with respect to basic audibility, familiarity to preschool children, and speech sound distribution. For preschool children colored pictures corresponding to the stimulus word were used for response material. For older children mimeographed black and white reproductions of pictures were employed with space for written response. Each stimulus word was preceded by a three-word carrier phrase. Three parallel series of 12 stimulus words were used, the intensity of each stimulus except the tenth being attenuated by three decibels, the ninth and tenth being equal in intensity. Children who failed to respond correctly to the ninth and tenth stimulations in any one of the three series were considered below the mean of their age group.

Systematic testing and retesting on 432 kindergarten and primary grade ears and 286 sixth grade ears was employed to determine the reliability of this test.

HILL

Unilateral Deafness Following Latent Otitis

Everberg, G.: Acta Otolaryngol. 45:4 (July-Aug.) 1955.

A study of 70 cases, 7 to 21 years of age, with total unilateral deafness, revealed, in addition to the deafness, absence of vestibular function and x-ray signs of labyrinthine damage on the same side in 15. Otoscopic examination showed scarred membranae tympani in 13 of these. While mumps has long been known to be an etiological factor in total unilateral deafness, the possibility of other causes, such as meningitis, trauma, heredity, labyrinthine aplasia and "perhaps more frequently otitis which has escaped detection" must be considered.

HILL

Vestibular Function in Deafness and Severe Hardness of Hearing

Cernvig, J.: Acta Otolaryngol. 45:4 (July-Aug.) 1955.

Vestibular studies, using rotation and caloric tests were carried out on 486 children, all in schools for the deaf, in an endeavor to ascertain if this could be of value in determining the etiology of the hearing impairment. Normal vestibular function was found in a great majority of those with severe inherited hearing loss. Abolished or abnormal responses were noted in those suffering from sporadic recessive deafness, a number of whom also had retinitis pigmentose. Eighty per cent of those of prenatal or natal origin showed normal responses confirming the biological rule that the more primitive organs are more resistant than those of later development. Abolished vestibular function suggests hearing loss acquired after birth. In eight per cent of all cases there was lack of uniformity between rotation and caloric response. As syphilis could be definitely excluded, it was considered that this finding could no longer be considered as diagnostic of luetic neurolabyrinthitis. It was felt that the majority of cases in which a careful history fails to reveal the probable cause were due to genetic factors.

HILL

Hearing and Speech in the Case of Partially Deaf Children

Torres Gassó, J. M., and *Perelló, J.*: Acta Oto-rino-laringol. Ibero-Amer. 7:272-295, 1956.

Partially deaf children frequently suffer the disadvantages of being considered to be deaf-mutes and therefore incapable of audible communication. These children can be trained to speak normally and to take their place in normal society if the degree of hearing loss can be determined by audiometric tests and improvement can be brought about by hearing aids. If the hearing loss is so great that the frequencies used in ordinary conversation are not audible, training in lip reading and in articulation is valuable in helping the child to adapt himself to a normal environment. Speech training is difficult in these cases because the child has no auditory image or memory of words, and also because of "neurologic immaturity."

A normal infant perceives sounds at the age of two months, and localizes them at five to six months. A child who does not respond to a familiar voice at the age of a year is either completely deaf or seriously deficient mentally. A partially deaf child reacts to loud voices or to sounds by reflex movements of the eyes, changes in expression, movements of the fingers, etc., but does not localize them correctly. The degree of hearing of infants too young to be tested by ordinary audiometric methods can be determined to some extent by mechanical devices which emit a wide band of frequencies, or by psychogalvanic stimuli to which the infant reacts by palpebral, oculomotor, and facial reflexes. One useful test in the case of small infants is electroencephalography. While the child is in a drowsy state induced by the electrodes, auditory stimuli are applied. Any response to the sounds is recorded by the waves on the encephalogram. The "K-wave complex" indicates stimulation of the temporal lobe. The stimuli begin with high tones at an intensity of thirty decibels. Progressively lower tones are used, at the same intensity. If the K-complex does not occur, indicating an auditory reaction, the intensity is increased to sixty decibels, or later to ninety decibels, and the frequencies are tested as before, to determine the hearing range, if the infant is not completely deaf.

In the case of children from five years of age on, the audiometric tests are supplemented by tests to determine the mental age of the subject, by means of toys suited to various grades and ages, some of which emit sounds. The child is given vocal instructions in the use

of the toy, and the response to the voice is observed. A bell or drum may also be used for the test.

Partial deafness produces changes in timbre, continuity, resonance, intensity, modulation of the voice, and in the respiratory rhythm and the adduction and vibration of the vocal cords. Speech tends to be explosive instead of rhythmic. Consonants are poorly heard, and are consequently not correctly spoken by the partly deaf child, and vowels are often confused. A is more clearly articulated than I and U. The voice is usually nasal or strident, and modulations of speech and accent are absent.

In re-educating the child to speak, the appearance, sound, and meaning of the object examined are associated with the word. Various methods of amplification of the sound are employed. The mother's voice, music, or sounds of cars, trains, animals, door closing, etc., are amplified, and identified by the child. Amplification of spoken words, by microphones, may be combined with labial training by means of mirrors and imitation of the teacher. The method varies with the mental age of the child and with the degree of deafness. It is essential that the child have confidence in the parents and teacher, and that there is understanding and affection, so that the child is integrated into the family, school, and environment in general, and does not have the feeling of being set apart and unable to communicate. A suitable hearing aid is essential when the child enters school. He learns by imitation of the speech of the other children. He should be assigned to a class according to his mental, rather than his chronologic age. The occupation or profession for which the child should be trained must depend upon the degree of deafness, his ability to adapt himself to his surroundings by means of a hearing aid and lip reading, and also upon his mental level.

HIGBEE

NOSE

New Aspects of the Secretory Function of the Nasal Fossae

Pizarro, F. R., and Hazen, E. C.: Bol. Espan. Otorrinolaringol. Bronco-esofagol. 8: 5-18, 1956.

The secretory function of the nasal mucosa is centered in the cup-shaped mucous glands and in the seromucous glands imbedded in the corium, in contact with erectile formations, which are activated

by the autonomic nervous system. The parasympathetic fibers increase the secretions and circulation. The sympathetic fibers act as antagonists.

Normal mucus is tenuous, clear, and transparent, and forms a thin film covering the mucosa of the nares, sinuses, pharynx, esophagus, and trachea. It is made up of mucin (2 to 3 per cent), water (97 per cent) and small amounts of sodium, calcium and potassium salts. When the nasal epithelium is exposed to toxic conditions the first symptom is a cessation of the secretion of mucus. Later the glandular cells become necrotic, and may be completely destroyed by lymphocytic and leucocytic infiltration.

The amount of nasal secretion depends upon the temperature and humidity of the air. Secretion is reduced when the humidity reaches 60 per cent. Hypersecretion at low temperatures is attributed to vaporization, which alters the osmotic pressure. Inflammatory changes in the nose and sinuses alter the PH and the mineral balance. Edema increases the mineral content whereas inflammatory conditions do not.

The PH of normal nasal secretion is neutral or slightly alkaline. The more alkaline the higher the bacterial count. In chronic rhinitis the reaction is usually neutral, and the mucus is sterile in 50 per cent of cases. In noninfectious, traumatic rhinitis, the reaction is neutral or slightly alkaline. Cold increases alkalinity, warmth produces acidity. Allergies and emotional factors may alter the PH. The nocturnal PH is lower than that during the day.

In rhinitis and sinusitis the calcium in the nasal secretion in situ was found to increase in proportion to the degree of inflammation, and the reaction became acid. The PH of normal secretion in vitro is neutral at first, later becoming acid, within twenty-four hours. The calcium also increases. The authors suggest that the change to the acid side may be due to an unknown biochemical catalytic substance.

HIGBEE

LARYNX

The Role of the External Laryngeal Muscles in Length-Adjustment of the Vocal Cords

Sonninen, A. A.: Acta Otolaryngol. Suppl. 130.

This investigation was carried out on 76 thyroidectomized patients, together with experimental work on cadaver material. Phone-

atric studies were made immediately before or as soon as possible after operation. It was found that when the pretracheal musculature (sternothyroid, sternohyoid and omohyoid) was sectioned bilaterally the pitch of the singing voice was lowered and the range narrowed. The same effect was noted but to a lesser degree when only one side was sectioned. These changes occurred independently of any interference with the cranial or caudal laryngeal nerves. Stimulation of the sternothyroid muscle alone with faradic current resulted in no change of pitch except when the head was bent backward.

Lateral roentgenograms have shown that the vocal cords lengthened as the pitch was elevated. In the cases whose pretracheal muscles were sectioned there was impaired lengthening associated with decreased pitch range.

The experiments showed that the pretracheal muscles participate in tensing the vocal cords. Lengthening the cords is dependent upon contraction of the entire pretracheal musculature. The sternothyroid muscle has a dual function, lengthening the cords for high tones (in conjunction with the other muscles) and shortening the cords for low tones.

The important function of the external laryngeal muscles, in synergism with the cricothyroid, is the lengthening or thinning of the vocal cords, thereby assisting in the production of high tones.

HILL

Melanosarcoma of the Larynx, and Other Matters

De Juan, P.: Acta Oto-rino-laringol. Ibero-Amer. 7:297-308, 1956.

Melanosarcoma of the larynx is extremely rare. In the author's experience, of 351 cases of cancer in this location, only two were sarcomas. According to Malinié, of 848 sarcomas in various sites, only one involved the larynx.

Sarcoma may attack either young or old individuals, but is usually observed in mature subjects. In 10 per cent of cases the patients are women.

Sarcoma of the larynx, whether primary or secondary, intrinsic or extrinsic, simple or mixed, can be classified as: fusocellular sarcoma,

round-cell sarcoma, giant-cell sarcoma or myeloplaxoma, or fibrosarcoma. In order of frequency the mixed tumors are: adenosarcoma, myxosarcoma, chondrosarcoma, angioadenosarcoma, rhabdomyoma, and, least common, melanosarcoma. Intrinsic cancers of the larynx which are walled in by cartilage are most effectively removed by surgery. Sarcomas situated in the upper portion of the epiglottis and the laryngeal wall show the least tendency to metastasis, while those involving the lingual wall rapidly invade the base of the tongue or the pharynx. Extrinsic tumors affecting the ventricular band invariably invade the base of the epiglottis, which has several orifices, favoring simultaneous involvement of the lingual and laryngeal surfaces.

The sarcomas, especially the fusocellular type, are yellowish in color. Melanosarcoma is bluish black. Sarcoma can be differentiated from epithelioma because of its rapid development, although this is not an infallible criterion. Sarcoma rarely results in ulceration. Fusiform-cell sarcoma and fibrosarcoma do not, as a rule, involve the lymph-nodes. However, in the case of the melanoma observed by the author, two nodes were affected, and presented the characteristic blue-black appearance due to melanin. The patient had a small wart on his left arm. No direct connection could be demonstrated between this formation and the malignant tumor of the larynx, but some relationship may exist between laryngeal sarcoma and cutaneous manifestations, as several cases have been cited in the literature describing their coexistence.

Sarcomine has been used in treatment, or if this proves ineffective, eradication by roentgenotherapy, or radical surgical extirpation with forceps and laryngoscopy spatulas, or electrosurgery, has been employed. Other authors advise extirpation by the external route, by thyrofissure (laryngofissure), laryngectomy, either total or partial, or cordectomy, leaving the vocal organ intact unless it is directly involved by the tumor. This is advised in early stages of the condition. However, if the tumor is established, the author recommends total extirpation of the larynx and careful cleaning out of the lymphatic chains in the neck, submaxillary, submental, and supraclavicular areas. In some cases the extirpated nodes have proven to be inflamed but not metastatic. Only 30 per cent of the lymph-nodes in cases of epithelioma of the larynx have been found to be metastatic; therefore, opinions differ as to the necessity for radical removal. However, De Juan systematically removes the suspected nodes, and also recommends postoperative roentgenotherapy. It is more important to save the life of the patient than to conserve the vocal function and to risk later lymphatic metastases or recurrences in situ.

The case is cited of a man 72 years of age who had suffered from chronic bronchitis due to excessive smoking. He complained of dysphagia, dysphonia, and dyspnea which had continued for three months. Laryngoscopy revealed a blue-black tumor on the laryngeal wall of the epiglottis extending toward the left. Two lymph-nodes on the right side of the neck were enlarged, and there was a wart on the left arm which suggested the possibility of cutaneolaryngeal sarcoma. Treatment consisted of laryngectomy from above downward, and removal of the affected glands. The tumor was found to be a melanosarcoma.

HIGBEE

Laryngeal Surgery under General Anesthesia

Martí Valverde, M., Checa Sagra, M., and *Márquez Broncano, S.*: Acta Oto-rino-laringol. Ibero-Amer. 7:261-271, 1956.

The results of surgical intervention under local anesthesia, in cancer of the larynx, are not always satisfactory. For this reason, general anesthesia was employed in ten cases of laryngeal epithelioma. Partial or total laryngectomy was carried out. Anesthesia was begun forty minutes prior to operation with glucose solution (300 to 400 cc), fenergan (2 cc), largactil (2 cc), and venocaine (5 cc). At the time of intervention the patient was somnolent. Respiration was somewhat accelerated, there was slight tachycardia, and arterial pressure was reduced. Actual anesthesia was obtained and maintained with pentothal in six cases, kemithal in two, and with thiobarbital in one case. In the remaining cases a barbiturate and ether-oxygen were administered through a tracheal cannula prior to operation. The patient's reflexes were normal. This is important, as coagulated blood and accumulated secretions could be eliminated by coughing during intervention.

For half an hour to three hours after operation the patients reacted to stimuli, but remained asleep, in a state of amnesia, and then regained full consciousness. Blood-pressure returned to normal. In all but one case, in which intervention was prolonged to three hours, the duration was two hours and twenty minutes, on an average.

The postoperative condition was good in all cases. The authors conclude that general intravenous anesthesia, with or without inhalation, is preferable to local anesthesia, in these cases. Surgery is facilitated by the fact that the patient is quiet, and by the absence of blood in the operative field, due to the controlled hypotension. A further

advantage is the regulated respiration, which renders the anesthesia by inhalation possible and reduces the dosage of the drugs given intravenously. Intubation is usually advisable in the preparation for total laryngectomy, as tracheotomy entails the danger of contamination of the operative field. However, in partial laryngectomy, tracheotomy is indicated, to insure free respiration and also because the presence of a cannula would render proper examination of the lesion more difficult.

HIGBEE

MISCELLANEOUS

Studies on the Conditioning of Air in the Respiratory Tract

Ingelstedt, S.: Acta Otolaryngol. Suppl. 131.

This monograph of some 80 pages, together with an appendix of recorded data, is a carefully documented account of physiological studies on air-conditioning in the respiratory tract. The investigation was carried out with a view to overcoming the errors inherent in the sampling methods of previous studies on this subject. The monograph describes in detail the technique of continuous simultaneous recording of the changing temperature and relative humidity of the air within the respiratory airways. A thermoelectric micropsychrometer is introduced into the subglottic space by means of a cannula inserted through the cricothyroid membrane. This, together with a special thermal element inserted in the nasal vestibule, makes it possible to measure the temperature of the air expired in the larynx and at the nasal vestibule simultaneously, while respiratory volumes are determined by means of an electric pneumographic system. Comparative studies on conditioning in different environmental temperatures are done by rapidly transporting the subject into a cold chamber.

A total of 29 examinations were carried out in 16 healthy volunteers. The mean temperature in the subglottic space for inspiratory air on nasal breathing was 32.3° C, and for expiratory air, 36.4° C. Relative humidity for both inspiratory and expiratory air was 98-99. On oral breathing the inspiratory temperature was 30.5° C and the expiratory, 36.2° C. Relative humidity was 90 during inspiration and 99 at expiration. Respiratory frequency and minute volume were practically uniform during nasal and oral breathing. Simultaneous recording of the temperature of expiratory air in the larynx and nasal vestibule showed a lowering of 3.2° C in the latter as the

effect of cooling during passage through the nose. Sudden changes in environmental temperature, by transporting to the cold chamber, appeared to have little effect in the temperature and humidity of the air in the subglottic area.

Immersion of the feet of subjects in cold water had no influence on the temperature and humidity of the subglottic air, nor on the temperature of the expired nasal air.

It was estimated that inspiratory air was conditioned to about 80 per cent by the time it reached the subglottic area and that 20-25 per cent of heat and moisture was recovered by the mucous membranes during expiration.

Careful reading of this monograph is suggested for all interested in nasal physiology as it is quite impossible to do it justice in any abstract form.

HILL

Nystagmography. Recording of Nystagmus in Clinical Neuro-otological Examination

Aschan, G., *Bergstedt*, M., and *Stahle*, J.: Acta Otolaryngol. Suppl. 129.

This monograph of 103 pages describes in detail a technique of recording nystagmus, based upon the principle of displacement of the cornea-retinal potential in movements of the eye. A direct writing electrocardiograph equipped with a preamplifier is used as the recording apparatus. In tracing horizontal nystagmus the electrodes are placed at the outer canthi of the eye. In vertical nystagmus these are placed above and below the orbit. The patient is grounded by means of a third electrode placed on the forehead.

This method makes it possible to determine the direction, eye-speed, frequency, and amplitude of the nystagmus. The margin of error is stated as plus or minus 10 per cent. The application of this procedure in various neuro-otological conditions, based upon the studies of some 2000 cases, is discussed. The author states that its usefulness "lies in its ability to establish an objective sign of disorder of equilibrium, thus proving the vertigo to be of somatic origin."

HILL

Anosmia and Agnosia Presumably Resulting from Anoxia

Zilstorff, K.: Acta Otolaryngol. 45:4 (July-Aug.) 1955.

The author reports a case of permanent loss of smell and taste following a complicated obstetrical delivery, during which there were several periods of cyanosis. There also was aphonia lasting two days. It was considered due to anoxia following hemorrhagic shock.

HILL

Notices

LV FRENCH CONGRESS OF OTOLARYNGOLOGY

The LV Congress of Otorhinolaryngology will meet from October 22 to October 25, 1957, in the Grand Amphitheater of the Faculty of Medicine in Paris under the presidency of Professor Aubry.

The two official subjects for discussion will be Cancer of the Ear and Tinnitus.

For information please apply to the administrative secretary, Cabinet-Raimond, 17 rue de Buci, Paris.

AMERICAN LARYNGOLOGICAL ASSOCIATION

Copies of the Transactions of the American Laryngological Association are available for general distribution at $8.00 a copy. Please send request with check to:

Dr. Edwin N. Broyles,
Editor Transactions
1100 North Charles St.
Baltimore 1, Md.

ACTA OTOLARYNGOLOGICA

There is a complete set of the *Acta Otolaryngologica* available together with all its supplements (excepting a few of the earliest ones). The present owner may be reached by writing Professor Gösta Dohlman, Lund, Sweden.

UNIVERSITY OF ILLINOIS

The next Laryngology and Bronchoesophagology course to be given by the University of Illinois College of Medicine is scheduled for the period November 4 to 16, 1957. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

TULANE UNIVERSITY OF LOUISIANA SCHOOL OF MEDICINE

The three year residency in otolaryngology offered at Charity Hospital of Louisiana at New Orleans on the Tulane University of Louisiana School of Medicine service is designed to qualify the holder for the examinations of the American Board of Otolaryngology and the practice of all phases of otolaryngology and endoscopy.

Candidates must be graduates of a class A medical school and must have completed a minimum of one year of general internship. An additional year of residency in internal medicine or general surgery is desirable but not essential.

All work is under the direct supervision of members of the Tulane Department of Otolaryngology, who are also members of the Charity Hospital Otolaryngological Staff; they are available at all times for instruction and guidance. Basic sciences are offered throughout each year of the residency during the academic year. The resident also participates in the program of the Speech and Hearing Center at the Tulane University School of Medicine.

The hospital year extends from July 1 of one year to June 30 of the following year.

Applications should be addressed to the Chairman of the Department of Otolaryngology, Tulane University of Louisiana School of Medicine, 1430 Tulane Ave., New Orleans 12, Louisiana.

TEMPLE UNIVERSITY
SCHOOL OF MEDICINE AND HOSPITAL

Postgraduate Course in Bronchoesophagology, May 20-31, 1957; September 9-20, 1957.

Postgraduate Course in Laryngology and Laryngeal Surgery, November 4-15, 1957.

These courses are all to be given in the Department of Laryngology and Broncho-Esophagology, Temple University Hospital and School of Medicine, under the direction of Doctors Chevalier L. Jackson and Charles M. Norris. The tuition fee for each course is $250.00. Further information and application blanks can be obtained from Dr. Chevalier L. Jackson, 3401 N. Broad Street, Philadelphia 40, Pa.

UNIVERSITY OF ILLINOIS

The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly in Otolaryngology from September 30 to October 6, 1957. The Assembly will consist of an intensive series of lectures and panels concerning advancements in otolaryngology, and evening sessions devoted to surgical anatomy of the head and neck and histopathology of the ear, nose and throat.

Interested physicians should write direct to the Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Illinois.

AMERICAN ACADEMY HOME STUDY COURSES

The 1957-1958 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, which are offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Detailed information and application forms can be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 15 Second Street S.W., Rochester, Minnesota. Registrations should be completed before August 15.

AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION

The American Association for Cleft Palate Rehabilitation will hold its 16th Annual Convention at the St. Francis Hotel in San Francisco on Thursday, Friday and Saturday, April 24, 25 and 26, 1958.

The association is composed of medical, dental and paramedical specialists who are interested in the rehabilitation of persons with cleft lips and palates.

SOCIETIES OF MILITARY OPHTHALMOLOGISTS AND OTOLARYNGOLOGISTS

The Society of Military Ophthalmologists and the Society of Military Otolaryngologists will hold their annual joint dinner meeting during the meeting of the American Academy of Ophthalmology and Otolaryngology in Chicago in October.

Cocktails and dinner will be served in the private dining rooms of the Palmer House Hotel at 6:30 p.m. on Tuesday, October 15, 1957. All members, ophthalmologists and otolaryngologists on active duty, or retired, are invited to attend.

Reservations may be made at the time of registration for the Academy Convention. Other information may be obtained from Major Stanley H. Bear, 3810th USAF Hospital, Maxwell Air Force Base, Alabama.

AFFILIATE MEMBERSHIP IN PROFESSIONAL GROUP ON MEDICAL ELECTRONICS, IRE

In order to further encourage participation by life scientists in the application of electronic techniques to the solution of bio-medical problems, an affiliate grade has been added in the Professional Group on Medical Electronics (PGME), Institute of Radio Engineers.

The Professional Group on Medical Electronics invites biologists, biophysicists, physicists, physicians, and others in the life sciences to participate in PGME activities through the affiliate plan. Since the PGME affiliate will not be a regular Institute member, the fee for a Professional Group Affiliate has been appropriately reduced for those persons already holding membership in other professional societies. The affiliate will receive notices of local and national PGME meetings. In addition he will receive the PGME Newsletters and the PGME Transactions containing medical electronic papers of current interest.

Further information on The PGME affiliate plan is available from Mr. L. G. Cumming, the Institute of Radio Engineers, Inc., One East 79th Street, New York 21, New York.

OFFICERS

OF THE

NATIONAL AND INTERNATIONAL OTOLARYNGOLOGICAL SOCIETIES

VI International Congress of Otolaryngology

President: Arthur W. Proetz, M.D., St. Louis, U. S. A.
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International Bronchoesophagological Society

President: Dr. Theodor Hünermann, Düsseldorf
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Jose Gros, Havana
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.
Meeting: Sixth Panamerican Congress, Brazil, 1958 or 1959

American Academy of Ophthalmology and Otolaryngology

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American Board of Otolaryngology

President: Dr. Gordon D. Hoople, Syracuse, N.Y.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa

American Broncho-Esophagological Association

President: Dr. Walter Hoover, Boston, Mass.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3, Pa.
Meeting: San Francisco, Calif., May 21, 22, 23, 1958

American Laryngological Association

President: Dr. Harry P. Schenck, Philadelphia.
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American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Percy Ireland, Toronto, Canada.
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American Medical Association, Section on Laryngology, Otology and Rhinology

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President: Sam H. Sanders, 1089 Madison Avenue, Memphis 3, Tennessee.
Secretary-Treasurer: Michael H. Barone, M.D., 468 Delaware Avenue, Buffalo 2, New York.

Pacific Coast Oto-Ophthalmological Society

President: H. Leroy Goss, M.D., 620 Cobb Building, Seattle 1, Wash.
Secretary-Treasurer: Homer E. Smith, M.D., 508 East South Temple, Salt Lake City, Utah

The Society of Military Otolaryngologists

President: Captain William C. Livingood, United States Navy (MC)
Secretary-Treasurer: Lt. Colonel Stanley H. Bear, United States Air Force (MC)

Canadian Otolaryngological Society

President: Dr. G.M.T. Hazen, 208 Canada Bldg., Saskatoon, Sask.
Secretary: Dr. G. A. Henry, Medical Arts Bldg., Suite 328, Toronto, Ont.